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ACUTE CHOLECYSTITIS

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New Orleans, La.

Cholecystitis is a common disease. Although there is almost unanimity of opinion that noncalculous cholecystitis is best treated conservatively, there is considerable controversy concerning the best therapy for cholecystic disease associated with calculi. Because the results following conservative treatment of noncalculous cholecystic disease are quite satisfactory and because noncalculous cholecystitis seldom is associated with complications, we[1] are of the opinion that this type of cholecystic disease generally should be treated conservatively; i.e., nonoperatively. On the other hand, we are equally convinced that patients with gallstones, whether they have symptoms or not, should be operated upon because of the potential complications that may be caused by calculi. There are many who believe that the symptomless gallstone should be disregarded, because in many instances gallstones are found at autopsy in individuals who had no symptoms during life. Although this undoubtedly is a true statement, it is not an argument against prophylactic removal of the gallbladder and calculi before gallstones cause serious complications, such as common duct obstruction, acute cholecystitis, acute and subacute pancreatitis, and carcinoma of the gallbladder. Any of these are serious lesions and all are preventable by the prophylactic removal of the biliary calculi.

The present communication is concerned with the treatment of acute cholecystitis, which is indeed a serious disease and which is directly attributable to calculi, since few, if any, cases of acutec holecystitis occur without obstruction of the cystic duct by calculi. In a series of 468 cases of acute cholecystitis observed at the Charity Hospital, there was a mortality rate of 8.3 per cent. Although it must be admitted that the prophylactic removal of the gallbladder containing

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gallstones entails a certain risk, the risk is certainly lower than this rate, being approximately ½ of 1 per cent. Although there are few physicians who would argue against cholecystectomy for cholecystitis associated with calculous disease in patients less than 50 years of age, there are many who are of the opinion that such an operation in an individual over 50 is undesirable. We[1] are convinced from our own experience that although cholecystectomy in a younger person is necessary, it is even more imperative in an older person, because acute cholecystitis is a much more serious condition in persons past 60 years of age. In the present series, there were no deaths in 134 patients under 40, 11 deaths (5.5 per cent) occurred in 201 patients between 40 and 60, and 28 deaths (21.1 per cent) occurred in 133 patients 60 years of age and older. It is thus seen that calculous disease in persons past 40 and particularly in persons past 60 years of age is a real hazard. Although, as mentioned previously, we[1] believe that younger persons with calculous disease should have a cholecystectomy with removal of the calculi, it is even more urgent that this should be done in individuals past 50 years of age.

The best treatment of acute cholecystitis is prophylaxis; i.e., removal of calculi before the cystic duct becomes obstructed which is the cause of acute inflammation. Unfortunately, prophylactic treatment of acute cholecystitis is not uniformly practiced and for this reason many cases of acute cholecystitis develop. Here, too, there is considerable controversy concerning the best method of treatment. Although the general tendency in recent years has been toward early operation in the acute attack, there remains disagreement and uncerainty as to the indicated treatment in various stages of acute episodes. We[1] have been of the opinion for some time that because acute cholecystitis is the result of obstruction of the cystic duct, usually by calculi, the initial changes in the gallbladder are not produced by invasion of bacteria, but are on a mechanical basis. These consist largely of vascular changes, and true bacterial inflammation occurs only after a period of two to three days during which time the gallbladder becomes more susceptible to invasion by bacteria which can gain entrance to it readily through the rich lymphatic bed between the liver and the gallbladder. If the gallbladder is removed in the early phase before bacterial invasion occurs and when changes are entirely on mechanical and vascular bases, the mortality rate is low and the morbidity is minimal. On the other hand, there are some who believe that acute cholecystic disease, since it rarely progresses to perforation, should be treated conservatively until the acute manifestations have subsided and then that extirpation of the gallbladder should be performed. In an effort to clarify these and other questions, we[1] studied 468 consecutive cases of acute cholecystitis observed at the Charity Hospital in New Orleans during the 11-year period from July 1, 1940 to June 1, 1951. In 462 patients, the gallbladder was examined at operation and in 6 additional patients at autopsy only.

INCIDENCE

The age of the patients ranged from 12 to 87 years with a mean average of 50 years. Ten per cent of the cases occurred in patients under 30 years of age,

60 per cent in patients between the ages of 30 and 60, and 30 per cent in patients over the age of 60. The present study is divided into two time-periods: the first, from 1940 to 1945 and the second, from 1945 to 1951. It is of interest that in the second period the number in the older age group increased, whereas in the earlier period only 21 per cent of the patients were in the 60 to 90 year age group, and in the later period 32 per cent were in this age group.

Cholecystic disease primarily is a disease of women. Eighty per cent of our cases of acute cholecystitis were in women and 20 per cent were in men. Cholecystic disease also primarily is a disease of the white race. There were 200 per 100,000 adult nonobstetric admissions in the white race as compared with 106 in the Negro race. The rates by race and sex were as follows: white females, 306; nonwhite females, 168; white males, 99, and nonwhite males, 28. It is thus seen that the incidence is extremely low in the Negro male.

CLINICAL MANIFESTATIONS

Twenty-one per cent of the patients had no previous symptoms of cholecystitis or cholelithiasis. However, 30 per cent had symptoms over three years; 25 per cent had symptoms less than one year; 10 per cent from one to two years, and approximately 5 per cent from two to three years. The clinical picture was quite dramatic: 99.8 per cent complained of pain in the right upper abdominal quadrant and 98 per cent had tenderness; 76 per cent had leukocytosis; 74 per cent had pyrexia; 49 per cent had a palpable mass; 49 per cent had abdominal rigidity, and 23 per cent were jaundiced. Cholelithiasis was present in 92.4 per cent and choledocholithiasis in 11.5 per cent. There were no stones in only 7 per cent. It is likely that in many, if not all, of these patients stones had been present, but had been passed by the time the patient was operated upon. Generalized peritonitis was present in 5.6 per cent of the patients, and adenocarcinoma was encountered in 6 patients (1.3 per cent). This last group is significant, because they fortunately developed acute cholecystitis which necessitated operation and in this way carcinoma was detected as an incidental finding after the gallbladder was removed and while the lesion still was limited to the gallbladder. Carcinoma of the gallbladder can be diagnosed clinically only after it has extended beyond the gallbladder and has involved adjacent structures. In such instances, it is never curable. It is also because of the possibility of carcinoma developing in the gallbladder of patients with cholelithiasis that prophylactic removal of the gallbladder should be done in all patients with gallstones. Although in this series, adenocarcinoma occurred in only 1.3 per cent, it has been estimated that carcinoma of the gallbladder is a complication of cholelithiasis in approximately 5 per cent of the patients.

Gangrene of the gallbladder was observed in 24 per cent of the patients and empyema in 14 per cent. Both of these complications were highest in the patients in whom conservative treatment was attempted, but was abandoned because of progressive symptoms. Gangrene was relatively frequent (28 per cent) and empyema was infrequent (12 per cent) in patients who were operated upon promptly. In the entire group initially treated conservatively, gangrene occurred in 18 per cent and empyema in 18 per cent.

TREATMENT

Cholecystectomy was done in 426 (91 per cent) and cholecystostomy in 56 (12 per cent). Choledochostomy was done on 130 patients (28 per cent) and in 51 of these (39 per cent), choledocholithiasis was present. Choledochostomy with cholecystectomy did not increase the operative mortality rate, which was 5.9 per cent for cholecystectomy alone and 4.6 per cent for the combined operation. A high operative mortality rate of 14.5 per cent for cholecystostomy was due to the critical conditions of the patients in whom this procedure was done.

MANAGEMENT OF ACUTE CHOLECYSTITIS

In the entire series, 289 patients (61.7 per cent) were operated upon promptly; 173 (37 per cent) were treated by delayed surgery, of which 125 (26.7 per cent) were successful and 48 (10.3 per cent) were unsuccessful, and 6 (1.3 per cent) were not operated upon. The 48 patients in whom conservative therapy failed represented 10 per cent of the entire series, and in 26 per cent of those patients in whom conservatism was attempted initially, the disease progressed in spite of medical treatment and operation had to be performed as an emergency procedure. Among 83 patients seen within 72 hours of onset, initial conservative therapy failed in 30 (36 per cent). In 57 patients similarly managed and first seen 4 and 10 days after onset, failure occurred in only 7 (12 per cent). Failure occurred in 11 of 35 patients (31 per cent) in whom conservative treatment was attempted more than 10 days after onset.

During the second half of the period investigated, there was a moderate increase in the performance of prompt operation and a corresponding decline in delayed surgery. There was a particularly gratifying reduction in the incidence of conservative failures from 16 per cent to 7.3 per cent. This probably was in some degree due to conservative treatment being attempted less frequently in patients seen early and more frequently in patients seen between the fourth and tenth days.

There is very little difference in the postoperative morbidity rate in the two series of cases. However, as might be expected, the duration of hospitalization was significantly greater in patients with delayed operation. By the twentieth postoperative day, 76 per cent of the surviving prompt operative patients, compared with only 22 per cent of the delayed operative patients, had been discharged.

As mentioned before, the mortality rate in the series of cases was 8.3 per cent and 7.5 per cent for the operative patients. These are admittedly high, but significant improvement occurred during the second period studied. After adjustment of rates for differences in distribution of age, race, sex, and severity of disease, a drop from 12.1 per cent during the first half of the 11 year period to 6.4 per cent in the second half was observed. As mentioned previously, the age of the patient was a significant factor in determining the mortality rate. There were no deaths in the 134 patients under 40 years of age; a mortality rate of 5.5 per cent in the 201 patients between 40 and 60, and a 21.1 per cent mor-

tality rate in the 133 patients over 60 years of age. The case fatality rate also varied with the severity of the disease. These were divided into the moderately severe, the severe, and the very severe with respective case mortality rates of 3.6 per cent, 4.2 per cent, and 14.6 per cent.

There also was a striking difference between the observed case fatality rate in males (17.9 per cent) and females (5.9 per cent). Because there was a much higher proportion of older male patients, age adjustments were necessary. This reduced the difference to 12.2 per cent and 6.7 per cent, respectively. When there was an adjustment for severity of disease, there were case fatality rates of 11.7 per cent in males and 6.8 per cent in females.

Two hundred and eighty-five patients were treated within four days of onset with an observed fatality rate of 6 per cent; 138 were treated from 4 to 10 days after onset with an observed fatality rate of 12.3 per cent, and 45 per cent were treated over 10 days after onset with an observed fatality rate of 11.1 per cent. The respective case fatality rates adjusted for age, sex, and severity of disease were 6.2 per cent, 10.3 per cent, and 14.1 per cent.

When the case fatality rates were studied for the type of management as related to the duration of symptoms interesting differences become apparent. In patients treated within 72 hours of onset, the case fatality rate was somewhat lower with prompt than with delayed operation. However, among patients treated from the fourth to the tenth day of disease, the adjusted case fatality rate was almost twice as high in those patients treated by prompt operation as in those treated conservatively. No deaths occurred in a small group of patients seen after 10 days and operated upon promptly. The case fatality rate was high in a somewhat larger group first seen after 10 days when treated by delayed operation.

CAUSES OF DEATH

Of the causes of death, 36 per cent were cardiovascular; 36 per cent were due to infection; 13 per cent were respiratory, and 15 per cent were due to miscellaneous causes. In an evaluation of the fatal cases, it is evident that in 19 (49 per cent) of the 39 deaths, avoidable delay in operative treatment appeared to be the primary cause of death. It is believed that death in 10 patients was due largely to delay in admission of the patients to the hospital. In none of these instances was the patient admitted within 72 hours of onset and on admission cholecystic disease was very advanced, usually with peritonitis. In two additional patients, the disease had become very severe within 32 hours of onset of symptoms with perforation and generalized peritonitis in 1 patient. These developed so rapidly that earlier operation might have been life-saving. In 7 patients, death was due primarily to delay in surgical treatment after admission to the hospital. In 5 instances, the patients were admitted within 72 hours of onset and in 2 instances, within five days of onset. In 2 additional patients, death was due to mistaken diagnosis and absence of correct treatment. In some additional patients, delay in surgical treatment was due partly to diagnostic uncertainty.

DISCUSSION

The present study illustrates the danger of acute cholecystitis and emphasizes the importance of prophylactic removal of the gallbladder and gallstones before the complication of acute cholecystitis occurs. It emphasizes also the urgency for this procedure, particularly in older people because the risk of acute cholecystitis in patients over 60 years of age is particularly great.

Serious complications, such as empyema, gangrene, perforation, and peritonitis, may occur, especially if surgical treatment is delayed. The case fatality rate in patients seen more than 72 hours after onset apparently was of greater significance than those patients seen earlier. Careful analysis of the deaths revealed that 50 per cent might have been prevented if surgery had been employed early in the course of the disease. Certainly, if the gallbladder is removed during the initial phase of acute cholecystitis when symptoms largely are the result of obstructive manifestations and vascular changes, the risk is much less. Undoubtedly in many such patients treated conservatively, the acute inflammatory process will subside. On the other hand, in individuals in whom it does not subside the disease is a progressive one and the risk is considerably greater.

Attempted conservative management frequently was more successful in patients in whom it was attempted 4 to 10 days after onset of symptoms, but it failed in a relatively high percentage of patients seen within 72 hours of onset and again in patients seen more than 10 days after onset. It was also in these last two groups that the case fatality rates for delayed surgical management were higher than for corresponding prompt operative therapy. The adjusted case fatality rate with prompt surgical treatment in the intermediate 4 to 10 day period was higher, but not significantly higher than that for initial conservative treatment. Death seemed to be due more commonly to the advanced stage of the disease which demanded immediate operation rather than to ill-advised prompt operation on patients in whom a more conservative form of treatment could have been attempted. Nevertheless, operation in this intermediate period does seem hazardous and if advanced cholecystitis does not demand immediate operation, experience indicates that initial conservative management probably is successful and operation can be performed later with less danger. Cholecystostomy was necessary in a much larger group of patients in the intermediate 4 to 10 day period than in either of the other two periods.

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EXPERIMENTAL VASCULAR GRAFTS. IV

ARTERIAL HOMOGRAFT DEGENERATION[a,b]

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"It should be emphasized that continual laboratory work is essential if the technique and results of homografting are to advance".

—Brock[1] (1953)

The transplantation of homologous tissues and organs has been a focus of medical interest for many years. Of all the tissues and organs homotransplanted, only blood, cornea, cartilage, bone, and blood vessels have been utilized clinically with any degree of success. Although there are many controversies as to the ultimate fate of these transplanted tissues, it is generally agreed that most of them serve merely as nonviable frameworks over which host cells may infiltrate.

The cellular life span of *transplanted* homologous blood is short, approximately 1 to 120 days. The normal mechanisms of blood cellular destruction remove these homologous cells when they no longer are capable of proper function. Thus, the degenerated homologous blood cells are automatically eliminated, leaving no foreign cellular material to complicate the effectiveness of the original transplantation.

Cornea probably is the most unique of all tissues in its response to homotransplantation. Maumenee and Kornblueth[11] (1948), and van Heuven[23] (1953) have shown quite conclusively that a majority of the corneal stromal cells continue to live and do not show a massive degeneration or replacement at any time during the postoperative period. Medawar[14] (1952) stated that corneal grafts are "immunologically privileged". The avascular cornea deriving its nourishment from aqueous humour filtrate, rather than blood cells or blood proteins, is protected from any immunity reaction the graft might provoke.

Another avascular tissue, cartilage, may retain its cellular integrity for years after homotransplantation (Mowlem,[16] 1941). Lasken and Sarnat[10] (1953) have shown that cartilage exhibits predominately an anaerobic metabolism. These authors concluded, therefore, that cartilage is better able to withstand the hypoxic state during transplantation and the subsequent period of cellular

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[f] Damon Runyon Cancer Fellow.
[g] Professor of Surgery.

adjustment to the host. Although cartilage may function satisfactorily for long periods of time as a homologous graft, clarification is necessary as to its ultimate cellular fate.

Wilson[25, 23] (1947) (1951) has stated concisely the present concept of homologous bone transplantation. Homologous bone grafts serve three functions: "1. They have a catalytic function in that their presence promotes and influences the osteogenic reaction. 2. They serve as a framework (nonviable) or scaffolding to guide the invading elements of the host. 3. They serve as a local supply of calcium".

Whereas there still remains much to be learned about the final changes which may occur after transplantation of arterial homografts, both fresh and preserved, the early cellular fate of these tissues has been well documented, by Enderlen and Borst[4] (1910), Yamanouchi[27] (1911), Williamson and Mann[24] (1947), Swan, Robertson, and Johnson[22] (1950), Miller, Callow, Welch, and MacMahon[15] (1951), Parsons, Gerbode, and Cox[18] (1952), Sauvage and Harkins[20] (1952), and Graham[7] (1952). The pattern of cellular events after arterial homotransplantation is essentially as follows: 1. Rapid disintegration of the intima lining of the graft. The intima is replaced rapidly by ingrowth of fibroblasts from both ends of the host vessel. These fibroblasts soon differentiate into flattened endothelial cells to form a smooth luminal lining for the graft. 2. The smooth muscle cells of the media rapidly disintegrate and are replaced by in-

TABLE I

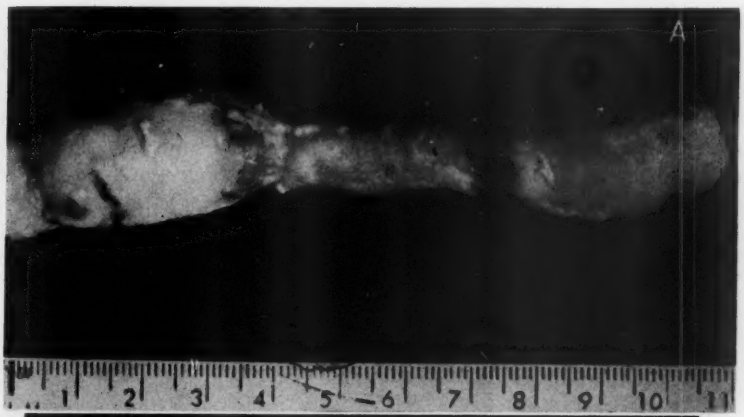
Thoracic aorta; short fresh arterial homografts*

Pig	Implantation (Days)	Per Cent Pig Weight Gain	Original Length of Graft (cm.)	Graft Calcification	Comments
21	79	0	2.55	No	Death due to peritonitis. Subendothelial yellow plaques.
37	35	0	4.5	Yes	Death due to pneumonia. Microscopic early medial calcification.
82	188	387	4.2	No	No microscopic calcification. Subendothelial yellow plaques.
83	186	301	3.7	Yes	Calcification in compact residual elastic tissue.
98†	276	937	3.7	Yes	2 large calcium deposits in vessel media. Mural thrombus adjacent to one of these.
126	226	578	1.2	No	Subendothelial yellow plaques.
131	268	728	0.6	Yes	Microscopic early medial calcification.
Average, 7 pigs	180	419	2.9	Yes, 57%	

* Graft length less than 5.0 cm. at implantation.

† See figure 1.

growth of fibroblastic connective tissue, again from the host vessel. 3. Elastic tissue degenerates, and becomes fragmented, but remains as a residuum of the original transplant for long periods of time. 4. This elastic tissue, probably because of loss of the intervening smooth muscle, becomes compact particularly toward the inner one-third of the media. 5. The original graft adventitia disintegrates and is replaced by host tissues. 6. Revascularization of the graft occurs primarily from the surrounding host tissues, but also to some degree from the host vessel ends. It is apparent that except for residual nonviable elastic tissue of the graft the transplant is replaced by host connective tissue scar. If the progression of events ended at this point, further study of blood



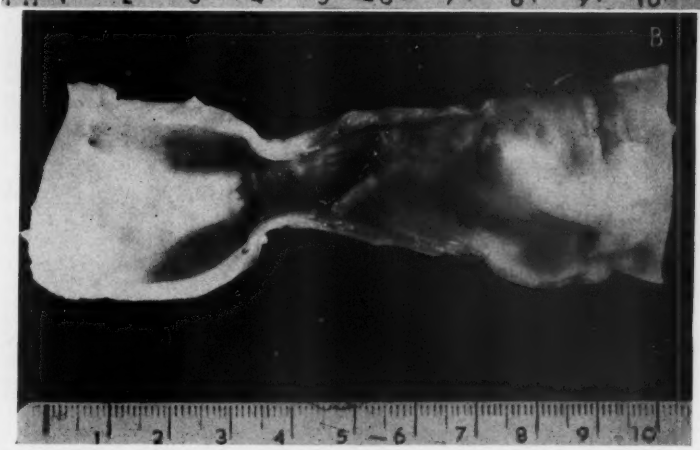


FIG. 1A. Inflated view of short fresh arterial homograft implanted for 276 days in pig 98. Proximal aorta is to the left.

FIG. 1B. Intimal view of homograft from pig 98. Note thickened wall of graft and the mural thrombus near upper anastomosis. This thrombus is attached to an intimal ulceration which is over a calcific plaque in the vessel wall.

vessel homotransplantation would not be necessary, as these uncomplicated, endothelial lined scar tissue tubes function well as conduits of blood. As will be indicated in the following experiments, there is a marked tendency for fresh and preserved arterial homografts implanted into the thoracic aorta of the growing pig to progress to a state of marked degeneration. The term *degenerative change* of arterial homografts as used in this presentation does not refer to the usual loss and replacement of graft cellular components but rather to the development of atheroma-like plaques or calcification in the homografts. The atheroma-like plaques observed are not believed to be identical with the atheromatous deposits seen in human vessels, but are thought to be indicative of graft degeneration.

METHODS AND PROCEDURES

Aortic homografts were implanted into the thoracic aorta of 41 healthy weanling pigs. The average weight of these pigs at the time of operation was 29 pounds (13.2 Kg.). The operative technic, postoperative care and the methods of graft processing were identical to those previously described by workers from this laboratory (Nyhus, Kanar, Moore, Schmitz, Sauvage, and Harkins[17] (1953)).

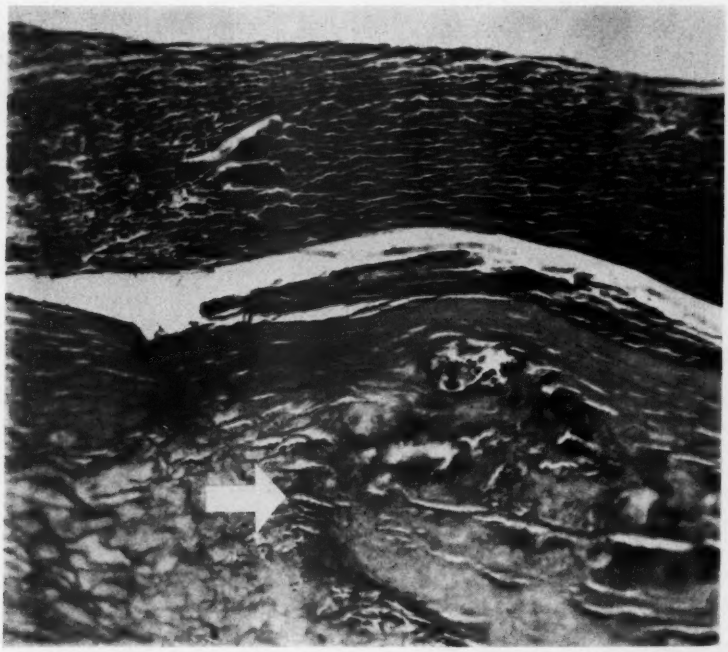


FIG. 1C. Thrombus (top of photomicrograph) loosely attached to intima of homograft from pig 98. Arrow indicates area of calcification in the vessel wall which is in direct relation to intimal ulceration and the mural thrombus (Hematoxylin-eosin stain. Enlargement ×100).

TABLE II

Thoracic aorta; long fresh arterial homografts*

Pig	Days Implanted	Per Cent Pig Weight Increase	Original Length of Graft (cm.)	Graft Calcification	Comments
19	56	58	7.1	Yes	Many subendothelial yellow plaques—Cause of death, pneumonia. Three small areas of calcification noted on microscopy.
43	193	451	10.1	Yes	Intima ulcerated over many of the calcified plaques in the vessel media.
44	87	187	6.25	No	Subendothelial yellow plaques. Death due to rupture of aorta above graft, at site of shunt ligature during operative procedure.
53	189	858	7.0	Yes	Many subendothelial yellow plaques. 2.5 mm. area of calcification.
72	258	354	5.5	Yes	One large area of calcification in elastic tissue of the media.
75†	295	841	9.4	Yes	Disseminated calcification of the vessel media.
76	8	—	6.25	No	No calcification on microscopy.
80	189	280	5.25	Yes	Calcific degeneration of the media with two associated ulcerations of the intima.
100	25	—	6.7	Yes	Two areas of early degenerative calcific change in elastic tissue of the media. Death due to empyema of both thoracic cavities.
101‡	321	1144	10.5	No	A perfect homograft. Microscopically appears as adjacent host aorta. No fibroblastic infiltration. Smooth muscle and elastic tissue intact.
102§	19	—	7.2	Yes	Death due to thrombotic occlusion at upper anastomosis. Residual smooth muscle apparent—Early calcific degeneration present.
109	252	896	9.1	Yes	Marked replacement of original graft by fibroblastic infiltration. Residual elastic tissue amorphous and fragmented. Calcification in this elastic tissue.
110	243	801	5.45	Yes	Marked breakdown of intima adjacent to calcification with superficial thromboses over ulcerations.
128	225	700	5.0	Yes	Marked medial calcification—Small aneurysmal outpouching midportion of graft.
132	267	652	5.6	Yes	Very thick subendothelial fibroblastic infiltration. Calcification is in direct continuity with this thickening, rather than more centrally placed in the media.
Average 15 pigs	175	502	6.8	Yes, 80%	

* Graft length greater than 5.0 cm. at implantion.

† See figure 2.

‡ See figure 3.

§ See figure 4.

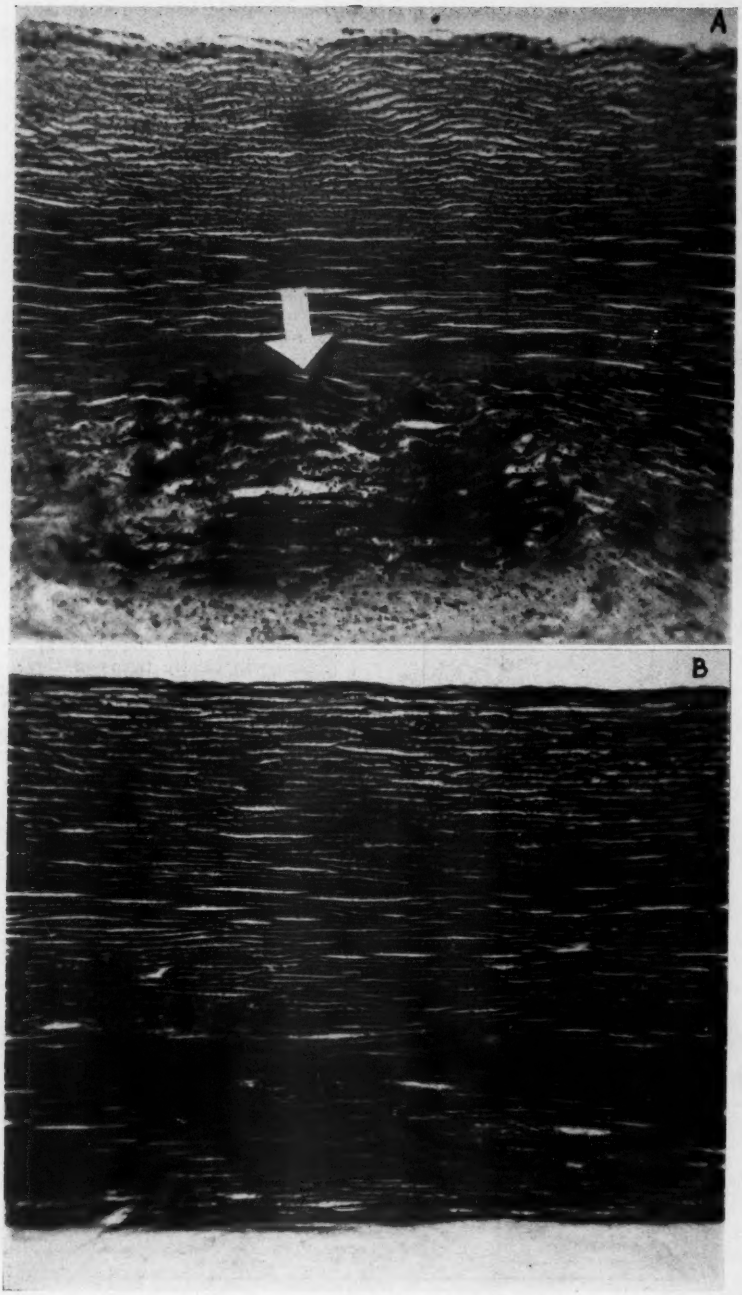


FIG. 2A. Photomicrograph of homograft implanted in pig 102 for only 19 days. The dark staining fibers indicated by the arrow represent areas of early calcification. The intima is at the top of the photomicrograph (Hematoxylin-eosin stain. Enlargement ×100).

FIG. 2B. Elastic tissue stain of homograft implanted for only 19 days in pig 102. Compare regularity and quantity of these elastic tissue fibers with those seen in figure 6D (Weigert's stain. Enlargement ×100. Intima is at top of photomicrograph).

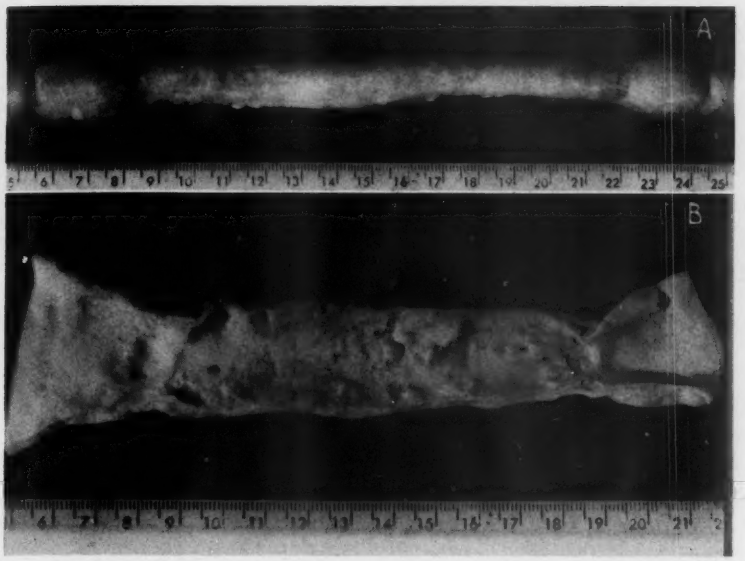


FIG. 3A. Inflated view of long fresh homograft implanted into pig 75 for 295 days. Proximal aorta is to the left.

FIG. 3B. Intimal view of graft from pig 75. Note the markedly roughened surface. Proximal aorta to the left.

Four types of arterial homografts were studied, short and long, fresh and preserved. An arbitrary definition of graft length was used in these experiments; those grafts over 5 cm. in length at implantation were defined as long grafts. A homograft was considered to be fresh when the graft was implanted into the recipient animal immediately upon its removal from the donor pig. A majority of the preserved grafts were stored in sterile light mineral oil at 4 C. Nutrient media (Tyrodes or Ringer's solution) was used to preserve 4 grafts. The 19 preserved grafts were stored from 1 day to a maximum of 413 days. The average period of preservation for the entire group was 106 days. Thirty-two of the 41 pigs lived to maturity, 6 to 10 months after graft implantation. Nine pigs died at varying intervals after operation. Three pigs died of graft thrombosis, 8, 19, and 106 days postoperative. The remaining 6 pigs died of the following nongraft complications: 3 of thoracic cavity infection; 1 of peritonitis; 1 of aortic rupture above the graft at a point of weakness in the host aorta created at operation by a heavy shunt ligature; and 1 of undetermined cause.

The average weight of the 32 pigs that reached full maturation was 228 pounds (103.6 Kg.). This represents an average weight increase in these same 32 pigs from operation until slaughter of 666 per cent.

RESULTS

The results of these experiments will be discussed separately under the four types of homografts implanted. These will include: (1) Short fresh thoracic

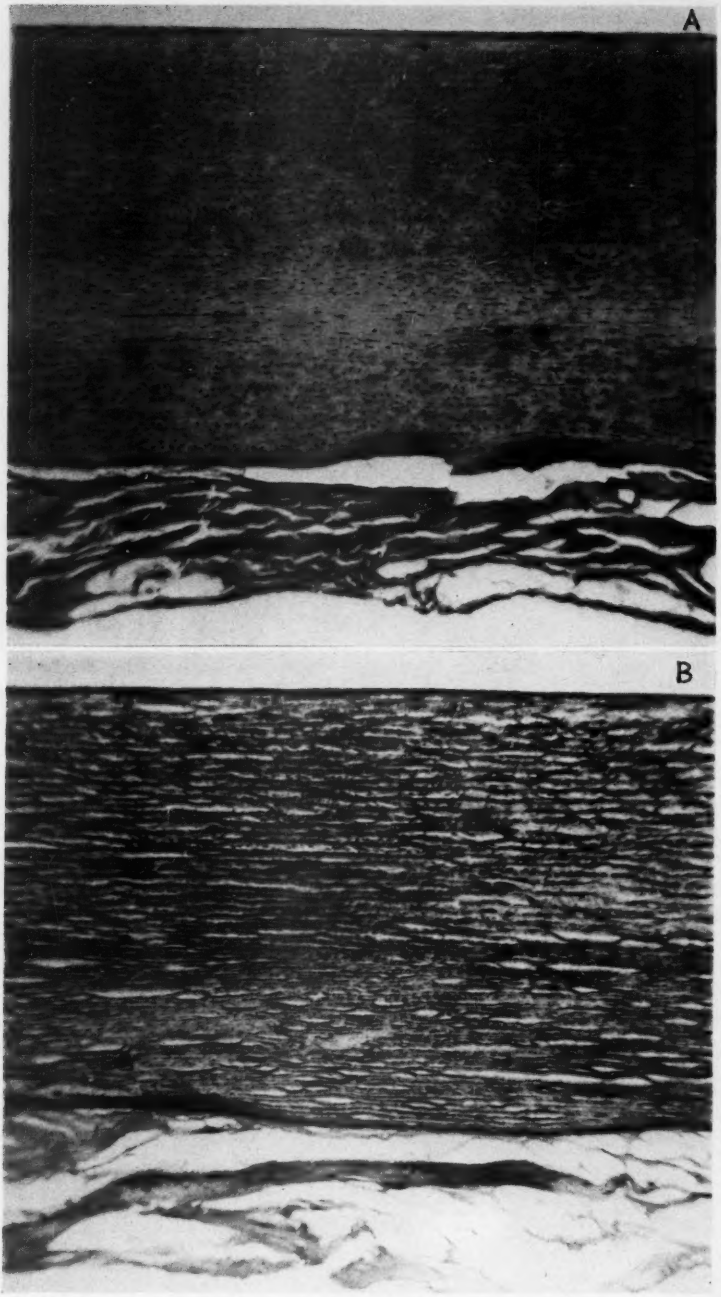


FIG. 4A. Photomicrograph of long fresh arterial homograft implanted in pig 101 for 321 days. Note the absence of connective tissue replacement of the graft (Hematoxylin-eosin. Enlargement ×100). Intima at top of photomicrograph.

FIG. 4B. Elastic tissue stain (Weigert's) showing uniform distribution of the elastic tissue fibers in homograft from pig 101 (Enlargement ×100).

aorta homografts; (2) long fresh thoracic aorta homografts; (3) short preserved thoracic aorta homografts; (4) long preserved thoracic aorta homografts.

1. *Short Fresh Thoracic Aorta Homografts* (Table I). There were 7 short fresh grafts implanted. Two of the 7 pigs died a relatively short time after graft implantation, 35 and 79 days. Both of these grafts had gross evidence of soft, pale yellow subendothelial plaques. Although a graft was implanted into pig 37 for only 35 days, it had microscopic evidence of early medial calcific degeneration. Two of the 7 grafts demonstrated gross calcification, but 2 additional grafts had microscopic calcification, and all 7 grafts had gross subendothelial yellow plaque

TABLE III

Thoracic aorta; short preserved arterial homografts*

Pig	Preservative Media—Stored at 4 C.	Days Preserved	Days Implanted	Per Cent Pig Weight Increase	Original Length of Graft (cm.)	Graft Calcification	Comments
17	M.O.†	243	225	838	2.4	No	Cicatrical band at lower anastomosis. No subintimal yellow plaques—No microscopic graft calcification
18	M.O.	247	106	119	2.3	Yes	Marked subendothelial fibroblastic infiltration. A rigid calcific tube.
45	M.O.	27	192	561	3.8	Yes	Endothelial ulceration associated with areas of marked medial calcification.
69	M.O.	125	219	677	1.1	Yes	Large areas of medial calcification.
90‡	M.O.	47	262	767	3.35	Yes	Complete shell of calcium, with ulcerated endothelial lining.
112	M.O.	84	258	1096	4.35	Yes	Multiple calcified areas in elastic tissue of media. Endothelium smooth and glistening.
125	M.O.	103	30	—	2.5	Yes	Early calcification of graft. The elastic tissue is only slightly disrupted. Cause of death—pneumonia.
130	M.O.	12	220	600	1.0	Yes	Very thick subendothelial fibroblastic infiltration. Elastic tissue compact next to this area of fibroplasia. Minimal calcification.
Average 8 Pigs	M.O., 100%	111	189	582	2.6	Yes, 87.5%	

* Graft length less than 5 cm. at implantation.
† Light mineral oil.
‡ See figure 5.

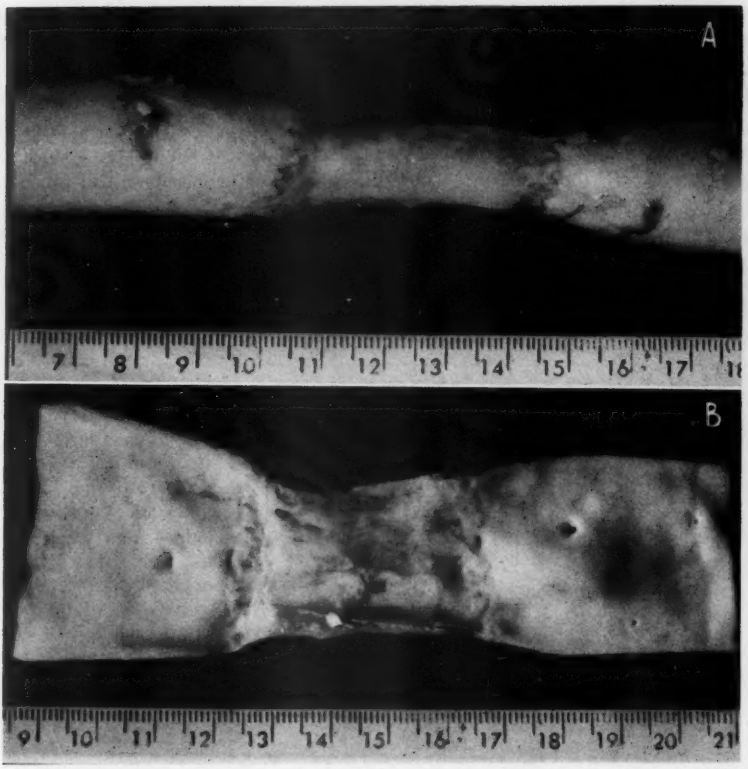


FIG. 5A. Inflated short preserved arterial homograft implanted in pig 90 for 262 days. This graft was preserved for 47 days in light mineral oil prior to implantation. Proximal aorta is to the left.

FIG. 5B. Intimal view of preserved homograft from pig 90. Note the layer of the calcium at the cut edge.

formation. The gross and microscopic picture of the graft implanted into pig 98 is shown in figure 1.

2. *Long Fresh Thoracic Aorta Homografts* (Table II). Fifteen fresh homografts over 5 cm. in length were implanted into growing pigs. Ten of these pigs survived to maturity. Five grafts examined 8 to 87 days after implantation had gross evidence of early degeneration with multiple atheroma-like subendothelial plaques. Although gross calcification was absent in the 5 grafts removed from pigs which did not mature, 3 of these grafts implanted for 19, 25, and 56 days had microscopic evidence of early calcification (fig. 2). Nine of the 10 remaining grafts were grossly calcified (fig. 3). The graft implanted into pig 101 was very unusual. This graft retained all of its cellular elements intact during the 321 days of implantation (fig. 4). This transplanted blood vessel appears to have maintained cellular integrity throughout the experiment.

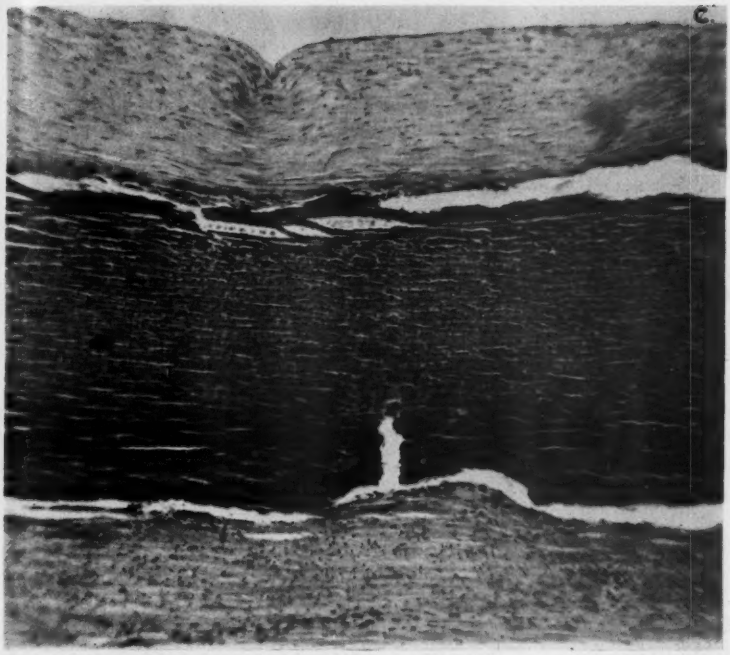


FIG. 5C. Photomicrograph of homograft from pig 90. There is marked subendothelial fibroblastic thickening. Calcification is prominent along both the external and internal aspects of the residual compact elastic tissue. Intimal surface at top of illustration (Hematoxylin-eosin. Enlargement ×100).

TABLE IV

Thoracic aorta; long preserved arterial homografts*

Pig	Preservative Media 4 C.	Days Preserved	Days Implanted	Per Cent Pig Weight Increase	Original Length of Graft (cm.)	Graft Calcification	Comments
20	RHS†	413	218	645	5.35	Yes	Four of the 11 grafts in this category were essentially solid calcium tubes, #36, 70, 133, 138. The remaining grafts had heavy deposits of calcium disseminated throughout the vessel media, particularly in the residual elastic tissue. Intimal ulceration was a common finding. Aneurysmal dilatation was not noted.
36	M.O.‡	1	187	671	8.6	Yes	
52	THS§	2	189	820	10.75	Yes	
70	THS	119	261	937	5.9	Yes	
71	M.O.	131	218	618	7.65	Yes	
73	M.O.	136	224	748	6.1	Yes	
79	M.O.	30	250	704	8.1	Yes	
81	M.O.	161	188	309	6.8	Yes	
84	M.O.	21	229	540	5.9	Yes	
133	M.O.	46	219	508	7.1	Yes	
138‖	THS	74	216	793	7.2	Yes	
Average, 11 pigs	M.O. 64%	103	218	663	7.2	Yes 100%	

* Graft length over 5 cm. at implantation.
† Ringer's homologous serum.
‡ Light mineral oil.
§ Tyrode's homologous serum.
‖ See figure 6.

3. *Short Preserved Thoracic Aortic Homografts* (Table III). There were 8 short preserved grafts implanted into growing pigs. Two pigs died prematurely, 30 and 106 days postoperatively. Both of these early grafts had apparent gross calcific degenerative change. Only one graft of the entire group failed to show either subintimal plaque formation, or calcium deposition. This graft was in pig 17. Although this graft had been preserved for 243 days at 4 C in light mineral oil, the only gross abnormality was a cicatricial intimal band at the lower anastomosis. The causative factor in the production of this band could not be determined. A representative graft from this group may be seen in figure 5.

4. *Long Preserved Thoracic Aortic Homografts* (Table IV). All 11 pigs of this group lived to maturity and all the grafts in this category had gross evidence of calcific degeneration. Complete replacement of the media by calcific deposits was present in 4 grafts, giving a gross appearance of rigid, partially endothelialized calcium tubes. Although 5 of the grafts were preserved for 1, 2, 21, 30 and 46 days, the graft end results were not improved when contrasted with those grafts preserved for longer periods of time. The graft implanted into pig 36 was preserved for only 1 day in light mineral oil, but degenerated into a

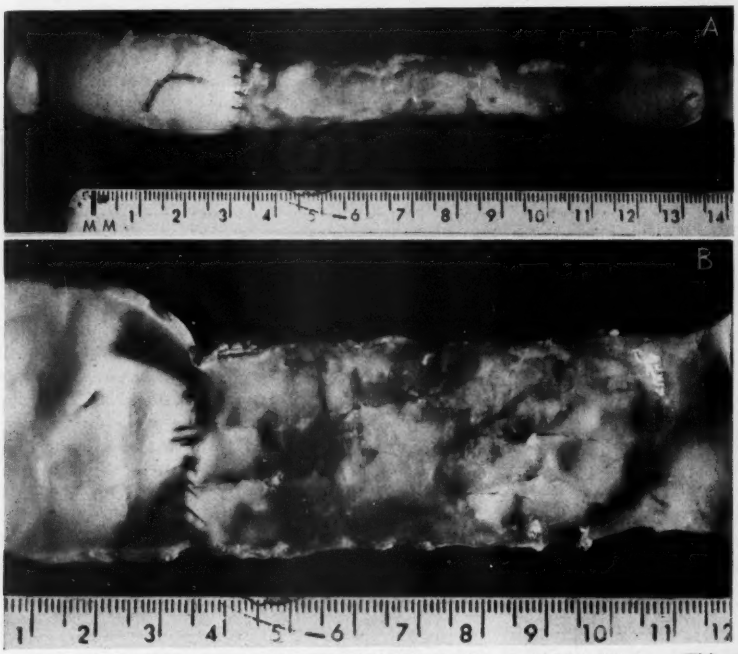


FIG. 6A. Inflated long preserved homograft implanted in pig 138 for 216 days. This graft had been preserved for 74 days in Tyrode's homologous serum. Essentially a rigid calcified tube.

FIG. 6B. Intimal view of preserved homograft from pig 138. The intimal cracks occurred when the graft was opened. Graft was extremely brittle.

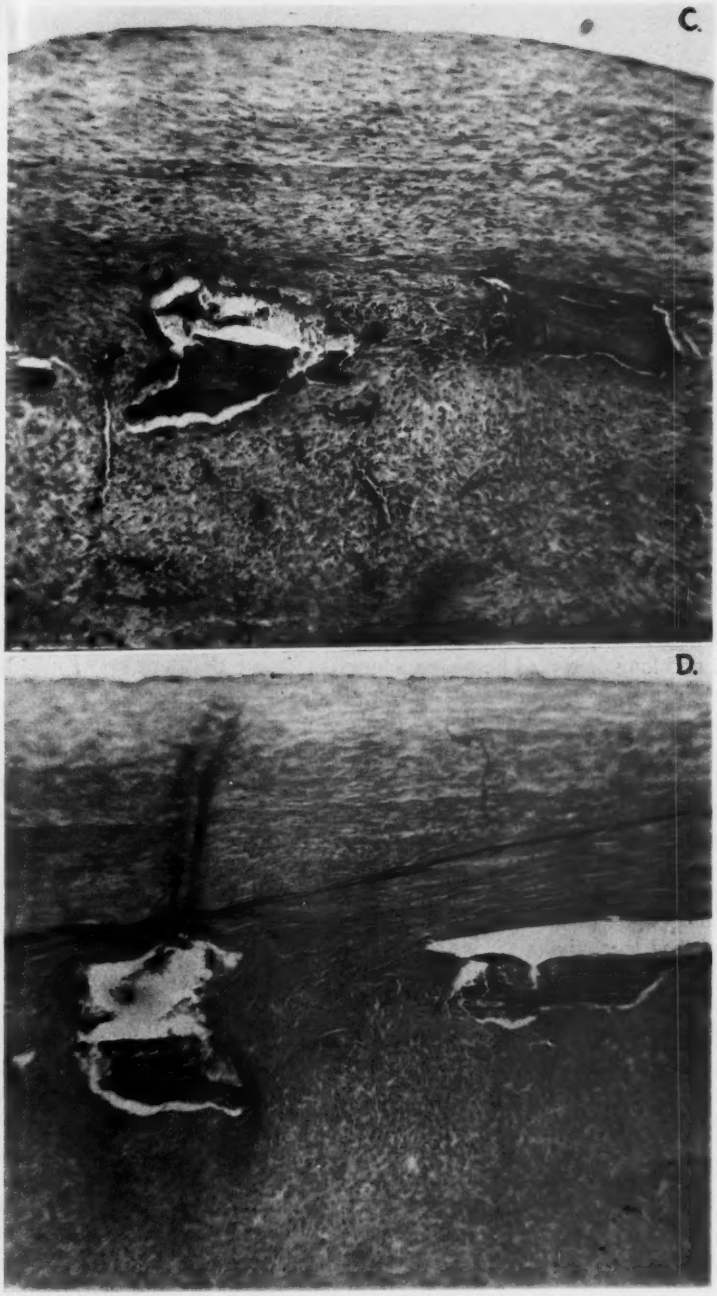


FIG. 6C. Photomicrograph of graft from pig 138. The graft has been completely replaced by fibroblastic infiltration except for the two dark areas in the center. These areas represent calcific deposition upon residual elastic tissue. Intima is at the top of the illustration (Hematoxylin-eosin stain. Enlargement ×100).

FIG. 6D. Elastic tissue stain (Weigert's) of homograft from pig 138. Note the minimal amount of elastic tissue present. Intima is at the top of the illustration. (Enlargement ×100.)

rigid calcific tube as mentioned above. Figure 6 demonstrates the graft implanted into pig 138.

DISCUSSION

The mechanisms by which vascular homograft degeneration takes place are not understood. From the data obtained in these experiments, the factors of graft length, anatomic site of graft implantation, graft preservation, and graft antigenicity will be considered as they may or may not be related to vascular homograft degeneration.

Vascular Graft Length. The shortest and longest fresh homografts implanted in these experiments were 0.6 and 10.1 cm. respectively. The homograft implanted into pig 101 (length at implantation 10.5 cm.) will be excluded from this particular section because of its unique response to transplantation. Both the short and the long fresh homografts which had been implanted for over 10 days demonstrated at least minimal evidence of degeneration denoted by subendothelial atheroma-like plaques. A majority of these same fresh grafts had either gross or microscopic evidence of calcific degeneration. Although the incidence of degenerative change was about equally distributed between the short and the long fresh homografts, the degree of degeneration—amount of gross calcium present per unit area—was somewhat greater in the longer grafts. When short and long preserved homografts were compared, the short grafts were found to be equally as degenerated as those over 5 cm. in length at implantation.

If the factor of graft length were of significance in the formation of calcific graft degeneration, one would expect to find a greater difference in the end-result than was noted in the short and long grafts of these experiments. We might assume that long arterial homografts are dependent upon nourishment from vessels originating in the cut ends of the host vessel, and that severe necrosis of the central graft may occur prior to revascularization. Attendant upon this assumption, we might expect calcification to be most prominent in the central portions of the graft. This was not our observation. The calcific plaques were distributed throughout the entire graft and seemed more severe near the lines of anastomoses. McCune and Blades[12] (1951) implanted very long (22 cm.) preserved aortic homografts between the thoracic and abdominal aortas of adult dogs. Degeneration of these grafts to a point of calcification was not noted. Subintimal yellow deposits were noted in those grafts which had been preserved longest. These deposits were found in the proximal portions of the grafts rather than toward the center of the transplant. McCune, Thistlethwaite, Keshishian, and Blades[13] (1952), by means of india ink injections of vascular graft transplants, were able to show that the major nutrition of at least the adventitia and the outer one-third of the vessel media was derived from perigraft host tissue vessels. Within one week after graft implantation they were able to demonstrate a considerable number of blood vessels entering the graft from the surrounding organs and tissues. These investigators were unable to demonstrate any vasa vasorum of the graft in continuity with the vessel lumen for supply of the inner portions of the graft, but postulated that nutrition filtered by some mechanism

through the new endothelium. Geiringer[5] (1951) stated that, "the depth of tissue which can be directly nourished by filtration from the main lumen depends upon the height of the filtration pressure and is therefore greater in the large arteries than in other blood vessels. In normal arteries this depth corresponds approximately to that of the intima plus the inner third of the media". From these observations it is apparent that nutrition is assured to most of the graft wall irrespective of graft length.

Anatomic Site of Graft Implantation. A majority of the arterial homograft experimentation has been performed utilizing the peripheral arteries and the abdominal aorta as the recipient sites for homologous artery transplantation. Arterial homograft degeneration (to calcification) has been alluded to in many experimental presentations as an *infrequent* late complication of homologous arterial grafting in the abdominal aorta and peripheral arteries. Parsons, Gerbode, and Cox (1952) noted heavy calcific deposition in short preserved arterial homografts implanted into the abdominal aortas of adult dogs. Only 1 of 6 such grafts was free of calcific degeneration. These results compare very closely to those observed in our thoracic aorta grafts. This essentially is the only reference made in the literature to such a high incidence of marked degenerative change in abdominal aorta arterial homografts. Three other references, including one from our laboratory, which are more typical of the results usually reported will be cited.

Gross, Bill, and Peirce[8] (1949) implanted 25 preserved arterial homografts into the abdominal aortas of adult dogs. Of the 17 grafts which had been inspected at the time of the report, only one had evidence of *intimal sclerosis.* These grafts were examined up to 6 months after implantation. Miller, Callow, Welch, and MacMahon (1951) found no gross or microscopic evidence of calcification in 29 fresh and preserved arterial homografts implanted into the femoral arteries of adult dogs when examined 16 days to 12.5 months after implantation. Sauvage and Harkins[21] (1953) found degenerative changes in only 2 of 41 fresh and preserved aortic homografts implanted into the abdominal aortas of growing pigs. It was this striking difference in graft end-result obtained by merely changing the anatomic site of transplantation which made us speculate upon the variables which may have been introduced.

What relationship the slight blood pressure differential between the thoracic and abdominal aorta, and the peripheral arteries may have upon the graft end-results should be determined. Kanar, Nyhus, Schmitz, Sauvage, Moore, and Harkins (1953) noted that the microscopic picture of these degenerated homografts is very similar to that of Mönckeberg's medial sclerosis of the peripheral muscular arteries. It seems hardly tenable that a correlation can be made between the *wear and tear* hypothesis for peripheral artery arteriosclerotic change and the early calcification seen in thoracic aorta arterial homografts. A corollary may be seen, however, in the early arteriosclerotic changes of hypertensive vessels proximal to a coarctation of the aorta. These hemodynamic factors of graft degenerative change now are being studied in our laboratory.

The difference in surrounding tissues in the abdominal and thoracic region (muscle, peritoneum, and pleura) may have some bearing upon the rate of re-

vascularization of these homografts. This is particularly true when comparing arteries in the extremity covered with vascular-rich muscle to the relatively avascular tissue available in the abdomen and chest. When comparing the relative vascularity of the retroperitoneal region of the abdomen with the retropleural area of the thorax, it appears certain that the former is more richly endowed with numerous minute vessels in the areolar tissues.

Furthermore, the factors of the fluctuating endothoracic pressure with normal respiration and coughing may exert some influence which produces the more rapid and more extensive degeneration of vascular homografts of the thoracic aorta. This factor is difficult to assess objectively and has not been investigated to the present time.

Since this high incidence of calcific and atheromatous degeneration in thoracic homografts of the aorta was unexpected, we failed to determine the blood calcium, phosphorus and cholesterol levels serially in our animals. It may be that removal and transection of ribs is sufficient stimulus for mobilization of calcium with its subsequent deposition in areas of persisting reaction and continuing fibroplasia, i.e., at the rib fracture sites and the arterial homografts.

Vascular Graft Preservation. The preservation of vascular grafts has long been indicted as an influencing factor in vascular graft degeneration. This probably is a just indictment. The short and long preserved grafts in these experiments had more advanced degenerative changes than was evident in the fresh grafts. The length of preservation could not be well correlated with the degree of calcification. The graft implanted into pig 36 was preserved for only 1 day in light mineral oil at 4 C, and was one of the most severely degenerated grafts examined. The graft implanted into pig 17 had been preserved for 243 days in light mineral oil at 4 C. After 225 days implantation, this graft was devoid of any gross or microscopic evidence of degenerative change. One of the 2 preserved aortic homografts which Sauvage and Harkins (1953) described as showing degenerative change is a good example of why length and method of preservation cannot be entirely indicted as the etiologic factors in graft degeneration. This particular homograft had been preserved in light mineral oil at 4 C for 101 days. Two other grafts from the same original donor vessel segment, preserved in the same bottle of mineral oil for 150 days and for 181 days, when implanted into 2 other pigs demonstrated no degenerative change.

Cellular viability of the graft at the time of implantation has been stressed as an important factor in the prevention of graft degeneration. The fresh grafts implanted in these experiments certainly were viable, yet, a high incidence of early and also late calcific degenerative changes was present in both the short and long grafts. Pate and Sawyer[19] (1953), in discussing the lyophilization of arterial homografts (after this process cellular viability is lost), thought that implantation of injured homologous living cells may be deleterious. The injured cells may "give off metabolites and toxic products of degeneration". From these few observations, it is apparent that at the present time we cannot be dogmatic in our thinking about the effects of graft viability and graft preservation upon vascular graft end-result.

Vascular Graft Antigenicity. Dempster[3] (1951) stated, "Blood vessels, I believe, are not antigenic". He qualified this statement by adding, "If they (blood vessels) are antigenic their specific antigens are not strong enough to evoke an immunity reaction of dimensions capable of effecting disintegration". The latter statement may be more the true picture of antigenicity related to graft degeneration.

Conway, Stark, and Joslin[2] (1953) have studied extensively homotransplantation of skin in mice. Using the transparent tissue chamber technic, they have described two phenomena which are thought to be prime factors in the failure of skin homografts. Thromboses of recipient bed vessels was the original observation. When the phenomenon of recipient bed vessel thrombosis was prevented with anticoagulants, "new vessels grew up to the homologous skin graft, and turned and coursed around the graft. No penetration of the new vessels into the graft was observed. New vessels arranged themselves about the periphery of the graft in concentric circles, giving the appearance of vascular lamellae". Kiehn and Glover[9] (1953) have shown a very similar process occurring after homologous transplantation of bone.

McCune, Thistlethwaite, Keshishian, and Blades (1952) believed that transplanted arterial homografts are not effected by this lack of revascularization from the host vessel ends, and as was mentioned previously, revascularization from the surrounding host tissues. Whether the failure of skin and bone homograft revascularization is due to physical, chemical, genetic or immune factors has not been determined. Whatever the basic factor may be that prevents revascularization of skin and some bone homografts, it must be attenuated with respect to arterial homografts.

Medawar (1952) failed to detect any specific *antibodies* in the presence of a homograft. Thus discussions of *host-graft sensitivity responses* and *host immune reactions* must be tempered by a lack of objective evidence for such biologic phenomena. And yet, one cannot unequivocally deny the possible importance of antigen-antibody mechanisms in vascular homografts.

Two arterial homografts observed in our laboratory tend to add credence to the hypothesis of host-graft sensitivity. Sauvage and Harkins (1953) reported the persistence of normal cellular architecture in a one day preserved arterial homograft implanted into the abdominal aorta of a growing pig. The donor artery was removed from a non litter-mate pig. Pig 101 in these experiments had a long fresh arterial homograft implanted into its thoracic aorta. Complete maintenance of graft cellular integrity was observed after 321 days of implantation. In these two grafts, the factors of length, preservation, and sites of implantation were dissimilar, yet each graft was maintained as an apparently viable structure. It would seem that these two grafts were accidentally implanted into genetically similar animals, completely precluding any host-graft sensitivity response.

The homograft implanted into pig 101 was obtained from its litter-mate. Glover[6] (1952) has shown that skin grafted to litter-mates will survive if done soon (within 3 to 4 weeks) after birth. Pig 101 was approximately 8 weeks old

at the time of graft implantation. Whether the factors of age and litter-mate relationship were of importance in the unique success of this homograft is not known. However, if we exclude pig 101 from this discussion, the success of the preserved *abdominal* arterial homograft described above cannot be discounted. This abdominal homograft was capable of maintaining its viability, not because of its short length, short period of preservation, viability at implantation, or abdominal site of implantation, but because the factor of host-graft sensitivity was not present.

We believe that vascular homografts are antigenic in nature. If the antigenic potential of arterial homografts were compared with other homotransplantable tissues, vascular grafts would fall within a zone of antigenicity somewhere between the minimal antigenic response elicited by cartilage and the maximal antigenic response elicited by skin.

SUMMARY

The degenerative changes (atheroma-like intimal plaque formation and gross calcification) observed in short and long, fresh and preserved arterial homografts implanted into the thoracic aorta of 41 growing pigs are presented. A high incidence of calcific degeneration was noted, irrespective of graft type, length or preservation. The marked degeneration of thoracic aorta homografts is in direct contrast to previous observations in similar grafts implanted into the abdominal aorta of the same experimental animal. The factors of graft length, graft preservation, anatomic site of graft implantation, and graft antigenicity are discussed as they may or may not be related to arterial homograft degeneration.

CONCLUSIONS

1. Fresh arterial homografts usually lose their cellular viability, and are replaced by host tissues.

2. Original homograft elastic tissue may be retained in the host replaced graft for an indefinite period.

3. Calcific deposition primarily is upon residual graft elastic tissue.

4. Microscopic evidence of calcific degeneration may be seen as early as 19 days after graft implantation.

5. Given the proper conditions, which are rarely fulfilled, fresh arterial homografts may remain viable, and will respond to transplantation in the same fashion as arterial autografts.

6. The short and long, fresh and preserved, arterial homografts used in these experiments, demonstrated an unusually high incidence of degenerative change.

7. Cellular viability at the time of graft implantation did not alter the high incidence of degenerative change in these grafts.

8. Graft preservation increased the severity of the degenerative changes noted. A definite correlation between the length of graft preservation and graft degenerative change could not be made.

9. Arterial homografts may elicit a modified antigenic response after transplantation.

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SURGERY IN THE MANAGEMENT OF UTERINE CANCER*

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Since Professor Wertheim described his technic of radical hysterectomy for cancer of the cervix there have been many gynecologic surgeons who have attempted to revive or modify the procedure in their efforts to obtain a better prognosis for the patient suffering with this disease. This is, of course, a reflection of the general dissatisfaction with the methods of therapy at hand. At the present moment a woman with cancer of the cervix has less than one chance in three of surviving more than five years. Think of it, that is a mortality rate of more than 66 per cent. Is it any wonder that Bonney, Taussig, Miegs, and other distinguished and technically capable surgeons have searched for another method of attack upon this killer of women, which until now has been treated primarily by irradiation, with generally unsatisfactory results? I am speaking now of what is considered adequate therapy, and I shall have more to say later in regard to inadequate therapy insofar as irradiation is concerned, and much more to say about what we should properly designate as *grossly* inadequate surgery by *grossly* inadequate surgeons.

In the instance of uterine cancer part of this apparent inadequacy of therapy lies not in the inability of radioactivity to kill cancer, but in its inherent nonselectivity; its inability to kill cancer which has extended beyond the limitations of the uterus without too severely damaging the vital structures so closely adjacent to it. It logically follows, then, that the clinical stage of the disease is the prognosticator. It follows also that the earlier the diagnosis is made and adequate therapy instituted, the better the prognosis. This apparently superfluous and perfectly obvious statement has a profound significance, and I should like to repeat it. The earlier the diagnosis is made and *adequate* therapy instituted, the better the prognosis. The inadequacies do not all lie, by any means, with the therapy or with the therapist, radiologic or surgical, but also with the diagnostician and with the patient herself. We all must assume our responsibilities as diagnosticians and lay educators. We all must utilize every means at our command to inform the public of the importance of periodic check-up examinations and continue to instruct the profession in the methods. When the patient appears in our offices or clinics, we must hunt out the cancer, and if we find none, we must continue to encourage the patient to return regularly. No early, easily curable, uterine cancer will come to us with a label on it. We must look for it with every instrument of our diagnostic armamentarium.

The surgical management of uterine cancer divides itself into four phases: preventive, diagnostic, palliative, and definitive. It is best to discuss the first two at one time for they frequently may be carried out together, and in many

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instances it is essential to rule out the presence of a carcinoma before a corrective procedure is undertaken, whether it be a linear cauterization or a complete hysterectomy. For instance, a simple cervical polyp may prove to be associated with malignancy. The carcinomatous change may be in the polyp itself, or cervical cancer and a benign polyp may occur in the same cervix. We have seen several patients with advanced cervical squamous cell cancer who gave a history of intermenstrual bleeding that originally was treated by removal of a microscopically benign polyp only several months previously. It is suggested that all patients, with what grossly appears to be simple erosion of the cervix, have the benefit of a Papanicolaou smear before cautery is done, and this only if she has had no irregular bleeding. If her clinical history is that of any type of abnormal discharge, she should have the cervix biopsied and the uterine cavity curetted before the cauterization is done. This is, of course, true particularly if the patient is over 35 years of age. It is my practice to make a Papanicolaou smear on all patients who have even the slightest bleeding and in whom a cervical polyp is found. This can be done at the time the polyp is removed.

The cytologic smear is nothing more nor less than a screening procedure. It is simple to make, but must be read concientiously by a competent cytologist. It should be made by aspiration and scraping, using two slides for each specimen. The material is placed upon one of each of two slides, the other one pressed against the corresponding slide, and then the two are slid apart. Both are placed in the fixative (alcohol 95 per cent and ether, equal parts). If it is necessary to mail the slides to the cytologist they may be air-dried after fixation, and mailed in a slide mailing carton. Staining is quite as satisfactory after this method as when the slides are kept moist with glycerine as was earlier recommended. The interpretation of the cytologist's report is of the utmost importance. A positive or suspicious smear demands pelvic examination under anesthesia, adequate biopsy of the cervix and curettage of the uterine cavity. A negative smear in the presence of symptoms means *absolutely nothing*. The symptoms must be explained.

Methods of cervical biopsy deserve some comment. There are two general methods of biopsy of the cervix. One is an office procedure and may be done with a biting forcep such as the Thoms[6] modification, or the endocervical coning biopsy instrument such as has been described by Gusberg.[2] The other is the cold knife biopsy. Admittedly, the latter requires hospitalization for an anesthetic is necessary. When the cervical biopsy is done, the endocervix and the endometrium also may be adequately biopsied and the internal gentalia carefully evaluated. If necessary, in early tumors, the entire cervix or endocervix may be removed as a biopsy specimen. Adequate and accurate biopsies cannot be made with the *punch biopsy* forcep, of which there are many varieties on the market. Another reason one should not rely on the punch biopsy is because it compresses the epithelial layer and gives rise to false positive diagnoses of carcinoma in situ. In my humble opinion, the reliability of the pathologist's report on tissue removed with a punch biopsy is inversely proportional to the number of different punch biopsy instruments that are available today.

The cold knife biopsy should be employed generously, together with frozen section, in any instance in which cautery amputation of the cervix (conization) is to be done. This also is true if there is the slightest clinical evidence of cancer of the cervix before total hysterectomy is undertaken. The importance of a preliminary dilatation and curettage also should be emphasized. The limitations of frozen section studies of the endometrium are well known, but nevertheless an adequate inspection of the cervix and curettement of the endometrium should precede any pelvic laparotomy in which it is the intent of the surgeon to preserve the uterus, or preceding any hysterectomy on a patient with a history of irregular bleeding or unexplained discharge. If the gross appearance of the curettings is at all suspicous, the major surgical procedure should await the pathologist's report on the permanent sections.

The importance of minor operations upon the cervix as a preventive measure should be re-emphasized. Linear cauterization to stimulate normal epithelization in simple erosion, cautery or cold knife conization of the chronically infected or leukoplakic cervix and reconstructive procedures on the lacerated and everted cervix all have an important place in the prevention of cancer. It is my belief, however, that complete hysterectomy is to be preferred to cervical amputation or plastic repair of the cervix if the patient is 40 years of age or older and has other lesser indications for hysterectomy, or has indications for pelvic laparotomy for benign adnexal disease, providing, of course, that child-bearing is no longer important to her.

Carcinoma of the cervical stump seen in The Ohio State University Tumor Conference represents 9.2 per cent of all cervical cancer, which is similar to that reported elsewhere. This is reason enough for further and louder condemnation of the subtotal hysterectomy. It seems to me no more justifiable than the removal of but one tonsil or the removal of the distal two-thirds of an appendix. Of course it is important to dissect the bladder from the upper vagina; of course it is important not to injure the ureter in placing the forceps on the cervical parametrium, but the operation is no more technically difficult to do than the supravaginal hysterectomy, and I believe that those who think it so should not be doing hysterectomies. The retained cervix is not of any value in supporting the vaginal vault. The same supporting structures that maintain the topography of the cervical stump will support the vaginal vault if properly utilized, perhaps better. In my experience, prolapse of the cervical stump is more common than prolapse of the vaginal vault. The cervical stump is not the keystone of the supporting arch of the vagina; it is the thorn in the side of the gynecologic cancer therapist.

The patient who presents herself with postmenopausal uterine bleeding offers a distinct challenge at times. I am in agreement with Brewer and Miller[1] that postmenopausal uterine bleeding should be defined as uterine bleeding occurring one year after the last menstruation. The management of the postmenopausal bleeder assumes its importance when we realize that in a collected series of reports of 4,181 cases, 56.8 per cent of the patients had a malignancy of the genital tract. It has been my practice to hospitalize all of these patients for cervical

biopsy and curettage except those in whom the pelvic examination is clinically negative and who are receiving estrogen therapy. In these patients, if the cytologic smear is negative, curettage is not done immediately unless the bleeding does not stop within two weeks following the cessation of estrogen therapy, or if it recurs. This method of management may be open to critisism because cancer can be the cause of bleeding in patients receiving estrogens. Extreme care must be used in the evaluation of the history of the bleeding and the pelvic findings in these patients if such a method is followed.

The layman is quite cognizant of the fact that postmenopausal bleeding is evidence of cancer until proved otherwise. This is good, but the laity and the profession as well are not so impressed with irregular bleeding during the reproductive age. The patient does not seek medical aid nearly so soon and without the utilization of adequate diagnostic measures too many of these patients fall more or less automatically into the class of functional bleeders. There are many proponents of thyroid, of steroid therapy, of vitamin therapy and a host of other things in the management of function bleeding, but these methods do nothing but lose time. The extreme importance of the time lost in these cases should be shouted from the housetops. All of this is further emphasized when we consider the number of women under 30 years of age with uterine cancer. At The Ohio State University Hospital Tumor Clinic this represents 7 per cent. (58 cases).

PALLIATIVE SURGERY

Under the heading of palliation we may properly discuss the indications for surgery other than those designed as a primary attack upon uterine cancer. This most often is true in the case of cervical carcinoma. It is a well known fact that 40 per cent of patients with this affliction die in uremia. The uremic death is an easy one, but death from ureteral block has been shown to occur[4] in the absence of carcinomatous tissue in the pelvis, and obviously has been due to scarring following irradiation rather than to the tumor. This mechanism must be considered in patients having demonstrable increase in the caliber of the ureters as they are followed by intravenous pyelograms. Especially is this true in patients having had primary irradiation therapy for clinical stage I and II tumors and more so, if the hydroureter becomes evident in the first two years of follow-up, providing clinical evaluation of the pelvis does not indicate recurrence of the carcinoma. If these conditions are present and evidence of distant metastasis is absent, we are justified in transplanting the ureters, preferably to the dome of the bladder with utilization of a bladder flap if necessary, or to the bowel or skin as the surgeon may desire and the technical problems dictate.

Irradiation reaction in the bowel at times has been a troublesome complication of the treatment of uterine malignancy. A low residue diet, a lubricant type laxative and antibiotics are indicated during roentgen therapy and in the postirradiation period of from 8 to 12 months. If, in spite of this, stenosis of the sigmoid or rectum subsequently occurs, temporary transverse colostomy is indicated. It should be emphasized that resection of the involved portion of the bowel should not be attempted until at least 15 months has passed since radiation ther-

apy was discontinued. By that time, in most patients, the reaction in the bowel will have subsided and a smaller area of stricture may be more easily resected.

The temporary colostomy also is a valuable *tool* in the management of recto-vaginal fistulas that are due to irradiation reaction, and it should always be done preceding any attempt to repair such a fistula. This will allow a complete resolution of ever present inflammatory reaction. In addition, generous biopsy of the marginal tissue must prove an absence of malignant cells. A considerable period of time should elapse following the completion of the irradiation which has caused the necrosing reaction and as much of the margin of the fistula must be removed as is feasible to re-establish the normal lumen of the intestine.

In the problem of urinary tract injury by irradiation there is no such convenient short circuiting operation as the colostomy in the case of bowel damage. Furthermore, the continuously wet patient, her family and her family physician put considerable pressure upon the surgeon to do something. *Herein lies the greatest pitfall.* The utmost patience and care must be practiced lest the patient fall into the hands of another surgeon who will do something, and much too soon. The bladder damaged by the sclerosing effect of irradiation will not heal unless sufficient time is allowed for re-establishment of blood supply. No attempt should be made to repair such a fistula under four to six months, and preferably six. Here again one must be sure that no cancer is present. It is believed that by far the best results in the repair of these fistulas can be made by the abdominal approach and, if necessary, by the transvesicle route, unless marked obesity or other medical contraindications dictate otherwise.

If the fistulas encountered primarily are due to, or associated with carcinomatous infiltration, one must choose between the diversion of the urinary or fecal stream and the more heroic pelvic exenteration procedure. If bladder or rectal involvement are associated with recurrent tumor growth, or with radioresistant tumors, the exenteration operation may be the method of choice. If, however, the patient first is seen in this clinical stage IV the urinary and/or fecal diverging operations should be coupled with radical radiation therapy. The general condition of the patient, of course, must dictate the method of attack, and in all of these cases one must assume that no matter what approach is made, it is made primarily for the comfort of the patient.

DEFINITIVE SURGICAL THERAPY

The combination of irradiation and surgery in the definitive attack upon adenocarcinoma of the endometrium is accepted widely in this country. Furthermore, the Annual Report on the Results of Radiotherapy in Cancer of the Uterus indicated that better results are obtained with intracavitary irradiation preceding complete hysterectomy and bilateral salpingo-oophorectomy than with surgery followed by deep roentgen therapy. We have accepted this method of treatment. None of the operable patients is given external irradiation. They all have received intrauterine irradiation with radium or cobalt 60 in doses which project an isodose curve at the outer surface of the uterus of from 5500 to 6000 roentgens. The location, extent, and type of tumor determine the variations in methods of treatment.

The clinical classification of Heyman,[3] or a modification of it, probably is the most useful in determining these variations in treatment. He divides the patients into the three classes: the clinically operable, technically operable, and inoperable. In the first class, the clinically operable, are included those patients in whom the tumor presumably can be removed and whose general condition is such that operation is deemed safe. In the second, or technically operable patients, are those who have a cardiovascular, renal or pulmonary complications which make surgery unsafe. Heyman also includes in this group the excessively obese patient. If, however, a combination of irradiation and surgery is the best method of therapy of carcinoma of the endometrium, then it is my belief that if obesity is the only complicating factor, excepting perhaps the technical ability of the surgeon, it should not preclude offering that patient a much better prognosis. The third class, or the inoperable tumors, are described by Heyman as those in which the cancer has extended into the parametrium or through the cervix into the vagina, or in which there are metastases outside the pelvis. In speaking of inoperability, he is referring to the simple complete hysterectomy because of the fact that regional lymph node metastasis usually is not to the ureteral, hypogastric and obturator groups, but to the lumbar nodes. There are patients, however, in whom parametrial extension may not yet be associated with lumbar node metastasis which are amenable to the radical hysterectomy and pelvic lymphadenectomy. This is particularly true of those patients in whom the tumor has extended into the cervix and has thereby involved the lymphatic drainage of the cervix. Ovarian involvement does not necessarily modify the operability of endometrial ancer if the lumbar nodes do not already contain metastatic elements.

The general plan of management of endometrial carcinoma is not so much modified by any classification as is the prognosis. The operability of these tumors is determined at the operating table and the involvement, gross or microscopic, of tissue above the pelvic brim should be the guide to operability. Unless the cervix is involved, or broad ligament extension is evident, the Wertheim operation is not indicated.

Adeno-acanthoma, carcinosarcoma and sarcoma of the endometrium are treated in the same manner as endometrial adenocarcinoma. Sarcoma botryoides and sarcoma of the myometrium, or that developing in uterine fibroids, has been treated by hysterectomy and bilateral salpingo-oophorectomy, with and without external radiation. Either method gives poor results.

Most cases of choriocarcinoma of the uterus are fatal because of the invasive character of the cells of this tumor and, therefore, its early metastasis. One must be free with the curet in any patient who expells a hydatidiform mole or who continues to have a bloody discharge for more than eight weeks following delivery. I have not included the postabortions in this group for it is my opinion that all patients aborting should have the benefit of a dilatation and curettage for this and other obvious reasons. Novak,[5] in a recent study of the cases reported to the Mathieu Memoral Chorioepithelioma Registry, found that 37.8 per cent followed abortion; 39.2 per cent followed expulsion of a hydatidiform mole for a total of 77 per cent, and only 23 per cent followed full term pregnancy. When the diagnosis of choriocarcinoma is made by curettage, the treatment of choice

is complete hysterectomy with bilateral salpingo-oophorectomy. This should be accomplished as radically as is found necessary at the time of operation. Because of the tendency for these tumors to quickly invade the parametrium, and because their malignant potentialities cannot always be determined under the microscope, and even though it seems hopeless at the operating table, as complete a removal as is possible should always be attempted. Irradiation following operation is not beneficial if the tumor seems grossly to be confined to the uterus.

The surgical attack upon carcinoma of the cervix has been extolled by some and damned by others. I do not intend to attempt to *sell* the radical Wertheim operation as the choice in the management of carcinoma of the cervix; however, a few remarks in its defense seem indicated at this time. Even though the operation is not new, it still is definitely in its infancy so far as the development of standard technics is concerned. It still is suffering intensely from growing pains, and because this is true, and because some haphazard attempts to use the procedure have produced poor results, it has given certain biased authors ammunition to use in their attempt to blast it into disrepute.

I do not believe that the radical hysterectomy is one that should be attempted by just any surgeon any more than I believe he should attempt a radical head and neck dissection, or a pneumonectomy, or a Whipple operation, or any other radical procedure unless he is trained and equipped to do so. The Wertheim operation should not be condemned because it is technically difficult; we do not condemn radical irradiation therapy. It, too, is technically difficult. If the radical hysterectomy with pelvic lymphadenectomy ever proves to give even slightly better results than irradiation alone, in stage I and II tumors, surgeons should train and equip themselves to do the operation, or the patients should be sent where it can be done. This, by the way, is the current practice in irradiation therapy: the patients are sent to the radiologists.

Another important consideration is that, at the present moment, irradiation remains the treatment of choice for carcinoma of the cervix. The surgical attack should be done only in a very well controlled series of patients, closely followed with the invaluable aid of a tumor registry system. Only by a fair and unbiased long time evaluation of the two methods can we determine which method, or combination of methods, is best.

The operability of a case of cervical carcinoma can almost invariably be determined prior to laparotomy. This involves careful pelvic evaluation by speculum, by vaginoabdominal, by rectovaginoabdominal, and by rectoabdominal examination. The proper clinical staging of the tumor will, in nearly all cases, determine automatically whether or not it is operable. The international classification is used by The Ohio State University Gynecologic Tumor Conference, and those patients who are considered for Wertheim hysterectomy are in clinical stage I or II.

Obviously, preoperative evaluation of the urinary and intestinal tracts must be made. Nonprotein nitrogen determination and excretory urograms should be coupled with cystoscopic evaluation of the bladder for evidence of edema or fixation. Proctosigmoidoscopy and barium studies should be made to evaluate

the colon. The general condition of the patient, of course, will determine which of these are clinically operable. Marked obesity, while it may not preclude simple hysterectomy, may at this stage of its technical development contraindicate the radical operation.

The chief surgical difficulty encountered in the procedure is in the preservation of the continuity of the ureters and bladder. If, inadvertently, the ureter is injured directly or the bladder opened, this does not present too great a problem. The real difficulty that arises is in the injury to the blood supply of these tissues.

The preservation of the superior vesicular artery is of the utmost importance if remote bladder damage is to be prevented. It usually is found arising from the inferior hypogastric artery a few milimeters distal to the origin of the uterine vessel. It also may arise as a branch of the superior vaginal artery. If this vesicular vessel is interrupted on one side, extreme care must be exercised so that it may be preserved on the opposite side. This is particularly true if intracavitary irradiation has preceded the surgery. The reaction to preoperative irradiation, in our experience, has caused more difficulty in regard to the bladder than to the ureter.

The arterial blood supply to the pelvic portion of the ureter is, derived from three sources. One of these, sometimes two, but never three may be sacrificed. The artery to the ureter which is derived from the bladder blood supply usually may be maintained, but at times is destroyed in freeing the ureter from its close association with the cervix and the vaginal vault. The ureteral branch of the uterine vessel usually is sacrificed when the larger artery is dissected off the ureter as the latter traverses the base of the broad ligament. The ureteral artery, which arises from the common iliac or from the inferior hypogastric just distal to the bifurcation of the iliac, must be identified carefully and preserved. If, however, it is inadvertently interrupted, the ureteral branch of the uterine artery must be maintained. This may be done by isolating the uterine artery and carefully dissecting it from its corresponding veins and from the ureter and ligating it just beyond the origin of its ureteral branch.

All of this careful preservation of the ureteral arteries may be for naught if the adventitia of the ureter is destroyed by too thorough stripping, or by too rough handling of this organ with toothed tissue forceps. I do not believe that the use of catheters in the ureters is of any particular help in the dissection. They even may be detrimental because it is easier to strip the ureter with a catheter in place and one may be carried away by his dissection. It has been my practice always to leave the peritoneum attached to the medial surface of the ureter as far down as possible.

The preoperative use of irradiation previously mentioned has been used in a small series of cases which were later subjected to radical surgery by me or my associate (John H. Holzaepfel). The details of this series will be reported elsewhere, but it is interesting to note that we encountered four fistulas in 30 patients so treated. In all of these operations the meticulous care of the blood supply, as outlined above, was practiced, but each of these individuals received preoperatively a total of 8000 roentgens or more to point A. There were three vesi-

covaginal, and one uretero-vaginal fistulas. Two of these had been previously treated with radium, receiving 10,560 roentgens and 8000 roentgens, respectively. One was treated with cobalt 60 and deep roentgen therapy, receiving to point A, 6000 roentgens from the radioactive cobalt, and 2280 roentgens. The fourth patient—the ureterovaginal fistula—received 8000 roentgens from cobalt 60. Since this has become evident, it has been our practice to give patients in whom surgery is contemplated, a total dose of intracavitary irradiation not to exceed 6000 roentgens to point A. Let it be said emphatically that previous irradiation therapy does not in any way make surgery more difficult. This knowledge has been gained from this series of patients and from many others operated upon following radium, cobalt 60 or deep roentgen therapy.

The results to date in this series of 30 patients may be briefly stated. All are alive and well from 1 to 10 years following treatment. The results may be tabulated further as follows:

L & W	*L & W*	*L & W*	*L & W*	*L & W*	*Total*
1 year	2 years	3 years	4 years	5 or more years	
5	3	4	2	16	30

Two of these were carcinomas of the cervical stump.

The lymph nodes which are to be removed are the ureteral or parametrial nodes, the obturator, the internal iliac, the external iliac, and the common iliac groups. It must be emphasized that the apparent node involvement or absence thereof, should in no way influence the thoroughness of the node dissection nor the abandoment of this part of the procedure. Firm, enlarged lymph nodes may be free of metastasis when examined microscopically, and small, soft, apparently uninvolved nodes literally may be full of carcinoma. It is for this reason that the node dissection is being done.

Amputation of the cervix and simple hysterectomy in the management of cervical carcinoma are mentioned only to be condemned. They have no place in its treatment except in the case of intraepithelial cervical cancer. If the biopsy has been deemed adequate and the diagnosis of carcinoma in situ has been made, complete hysterectomy is adequate therapy.

SUMMARY

Prevention of uterine cancer is a most important part of the surgical management of this disease.

Early recognition of uterine malignancy is the only means by which a respectable salvage rate of those afflicted can be obtained.

A combination of irradiation and surgery offers the best method of attack in endometrial carcinoma. This also may be true in the instance of cervical carcinoma. Much more evaluation is necessary to decide. In no event should surgery for carcinoma of the cervix be undertaken haphazardly, and to best obtain the proper knowledge of the value of surgery, it must be done in controlled and perfectly followed series.

Palliative surgery may be as radical as seems necessary to obtain the desired result. In selected cases, surgery may afford relief in ureteral block not due to

carcinoma. Reconstructive surgery must not be undertaken in the presence of tumor, and must await subsidence of irradiation reaction and control of inflammatory processes.

The results of treatment of stages III and IV cervical cancer and of advanced endometrial carcinoma are of little actual interest. These are neglected cases—neglected by the patient, the diagnostician or the therapist. Obviously, we must do all that is possible for them, but the real effort, both in money and physical endeavor, must be toward continuous education of the layman and the profession so that this neglect can be eliminated.

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POLYPOID LESIONS OF THE STOMACH

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The term polyp is derived from the Greek meaning *many footed* and was the name given to certain invertebrates of the cuttlefish family because of their method of attachment to rocks. Because of a resemblance to the invertebrate polypus, pedunculated tumors of varied types occurring in man have been referred to as polyps. Under this heading is included neoplasms, hyperplasias and even edematous formations such as nasal polyps. It thus becomes clear that any grouping of lesions under the heading of polypoid tumors of the stomach is based purely on the physical characteristic assumed by the growth rather than on any histologic characteristics. In fact, any histologic type of gastric tumor may assume a polypoid form. Since a clinical diagnosis must be made on either roentgenologic or gastroscopic observation, and the histologic diagnosis may not be apparent on these observations, the gross descriptive term polyp must be retained. A classification of polypoid lesions thus becomes much the same as for gastric tumors in general. The following classification modified from Richards[19] will serve as a general guide (table 1).

INCIDENCE

The incidence of polypoid tumors occurring in clinical patients is unknown and will remain unknown, since a large percentage of benign polyps remain asymptomatic. Most of the series of cases reported in the literature have been analyzed from the standpoint of benign gastric tumors versus gastric malignancy, or on the basis of what percentage of epithelial polyps are malignant or become malignant. These studies assume that most polypoid gastric lesions which are not papillary adenocarcinomas are benign in nature. Based on several excellent studies of autopsy material,[22, 3, 20, 23, 24] it would appear that approximately 20 per cent of all gastric neoplasms are benign and that most of these benign lesions are grossly of a polypoid nature. The incidence of benign polypoid tumors found at autopsy in the same series of cases varied from 0.25 to 0.8 per cent.

The relative incidence of benign gastric tumors is summarized in table 2. The four series cited comprise a total of 1761 cases studied and are quoted in most of the papers concerning this subject. The wide discrepancy presented by these four groups of cases can be understood easily when the papers are read carefully. Of the 560 cases collected by Eliason and Wright only 50 cases were from their own series, and of these 50 cases 74 per cent were reported to be of epithelial origin. Yet, by their own statement histologic studies had been made in only 16

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TABLE I

Classification of polypoid gastric lesions

	Benign	Malignant
1. Epithelial		
From gastric mucosa	Adenoma	Papillary carcinoma
Ectopic epithelium	Aberrant pancreas[17]	
2. Connective tissue	Fibroma	Fibrosarcoma
	Mixed connective tissue tumors	
3. Smooth muscle	Leiomyoma	Myosarcoma
4. Nerve tissue	Neurofibroma	Neurogenic sarcoma
	Neurolemmoma	
5. Fat	Lipoma	Liposarcoma
6. Vascular tissue	Hemangioma	Hemangiosarcoma
	Lymphangioma	
	Endothelioma	
7. Miscellaneous	Lymphoma	
	Dermoid cyst	
	Myxoma	
	Chondroma	
	Osteoma	
	Echinococcal cyst	
	Glomus tumor[9]	

TABLE II

Incidence of benign gastric tumors in percentages

1761 Cases

Type	Minnes & Geschickter[14]	Eliason & Wright[5]	Eusterman[6]	Thompson & Oyster[23]
Epithelial	35.2	21.0	63	59.5
Nonepithelial				
Myoma	36.6	57.3	26	23.3
Neuroma	10.9	—	2.8	8.5
Fibroma	4.5	4.1	3.4	7.4
Lipoma	3.4	5	.6	1.1
Vascular	4.3	1.7	3.4	—
Miscellaneous	4.6	7.6	1.7	—

of these cases. In the most recent series studied by Thompson and Oyster 66 of the 94 cases were from autopsy material with a great difference in percentage of types between the autopsy and clinical cases.

ETIOLOGY

The origin and development of mesenchymal polypoid gastric tumors presumably is no different from mesenchymal tumors elsewhere in the body. However, since Menetrier's[12] description of gastric adenomas in 1888, with the observation that these lesions were associated frequently with chronic gastritis, there has been speculation concerning the role of chronic irritation in the de-

velopment of these tumors. The fact that achlorhydria is present in over 90 per cent of patients with adenomas, and that atrophic gastritis has been demonstrated in the vast majority of stomachs removed for adenomas, suggests more than a coincidental relationship. The high incidence of gastric polyps and carcinomas in patients with pernicious anemia and achlorhydria also suggests a causative relationship between gastritis and polyps. Other exciting causes mentioned in the literature include intestinal parasites, alcohol, vitamin deficiencies and syphilis. McManus and Sommers[11] noted an unusually high incidence of gastrointestinal cancers in association with malignancies in various endocrine stimulated organs and suggests that the formation of gastric polyps may be influenced by hormonal imbalance including an increased secretion of pituitary growth hormone. The tendency for carcinoma to develop in a site away from a gastric polyp gives support to the concept of a general causative factor.[2, 11, 8]

CLINICAL SIGNIFICANCE

Although diagnostic and technical advances during the past decade have improved the cure rate in carcinoma of the stomach, the number of patients cured of gastric cancer remains discouragingly small.[15] Thus, the importance of premalignant or potentially malignant stomach lesions assumes great importance. Gastric tumors of mesenchymal origin evidently are of the same significance as soft tissue tumors elsewhere in the body. However, epithelial polyps arising from gastric mucosa are of much greater significance in view of the frequency of gastric cancer. The tendency of polyps of the colon and rectum to undergo malignant changes is so well known that the advisability of surgical removal of such lesions is accepted universally. While it is true that a high percentage of such lesions are within the diagnostic and therapeutic reach of the sigmoidoscope, the factor of poorer accessability to similar lesions of the stomach should not alter our therapeutic approach.

Review of the literature shows a fairly wide discrepancy in the ratio of benign to malignant epithelial polypoid lesions of the stomach (6 to 40 per cent).[4, 12, 21, 24] However, the potential malignancy of these lesions justifies Moyer and Clayton's[15] statement that "the really benign polypoid growths in the stomach are so rare that the possibility of their existence should never be used as the basis for deferring operative intervention." True malignant degeneration of a previously benign polyp is difficult to prove, although clinical case analyses are convincing.[24, 4, 21] The case reported by Klein and Geller[10] in which a patient had three similar polyps seen by gastroscopy, and two years later one of the polyps was found to be malignant, seems to offer conclusive proof to even the most skeptical. Our case no. 6 likewise offers strong supportive evidence. Despite the mass of evidence pointing to the potential danger of these lesions, there still are some advocates of the *nonoperative or observation* type of management of certain cases. Hay,[8] in a recent study, found size of the lesion the best criterion of diagnosis, finding only one malignancy out of 82 lesions under 2 cm. in diameter. The evidence presented by this author both as to difficulty in diagnosis

and potential malignancy of such lesions would hardly justify his final conclusion that "observational management of patients diagnosed as having small benign lesions is justified if they can be observed at frequent intervals." In a study of 85 polyps of the gastrointestinal tract Mikal and Campbell[13] found no relation between size of the polyp and malignancy. Paul and Logan[18] state that the most important aspect of gastric polyps is their tendency to become malignant, yet these authors still advise frequent observation by repeated gastroscopy for any suggestion of malignant degeneration.

CASE REPORTS

Case 1. H. H. M., a 67 year old white woman was admitted to the Kansas University Medical Center on April 28, 1948 because of gross hematemesis on the morning of admission. There was no history of previous gastrointestinal complaints. The only important physical finding was evidence of shock. The hemoglobin was 38 per cent. Transfusions of whole blood were administered. Upper gastrointestinal barium studies on the fourth hospital day were interpreted by the roentgenologist as "a large benign tumor that has become malignant". At operation nothing could be felt in the stomach, the bizarre roentgenologic picture apparently having been produced by clotted blood. A gastrotomy was done and a polyp 5 by 7 mm. in size was found on the anterior gastric wall. This was excised with an elliptical section of the gastric wall and proved to be a benign adenomatous polyp on histologic examination. The patient has remained free of gastric symptoms to date.

Case 2. D. R. J., a 73 year old Negro man was admitted to the Kansas University Medical Center on April 20, 1954 because of rectal bleeding and dizziness. There had been no complaints referable to the gastrointestinal tract except four episodes of rectal bleeding during the past six years. Physical findings were not important, except for evidence of an anemia. The hemoglobin was 6.1 Gm. He was treated with transfusions of whole blood, but continued to lose blood from the gastrointestinal tract. Barium studies showed a polypoid lesion in the fundus of the stomach (fig. 1). At operation a large nodular tumor was found on the greater curvature of the stomach and a subtotal gastric resection was done. The lesion measured 10 by 7 cm. in size and had an ulcerated surface showing two eroded blood vessels. Histologic diagnosis of submucous and subserous leiomyoma of the stomach was made.

Case 3. M. B. B., a 73 year old white man was admitted to the Kansas University Medical Center on Aug. 30, 1950 because of mild epigastric distress described as heart burn. He stated that his expectoration was streaked with blood on two occasions. The only important physical finding was auricular fibrillation. He had a hypochromic anemia with a hemoglobin of 10.5 Gm. Upper gastrointestinal roentgenologic studies showed multiple polyps of the distal portion of the stomach (fig. 2). A subtotal gastric resection was done. The removed specimen contained seven polyps along the anterior wall and greater curvature side of the gastric antrum. The histologic diagnosis was multiple adenomatous polyps with chronic inflammatory reaction.

Case 4. J. E., a 73 year old white man was admitted to the Kansas City Veterans Administration Hospital on Dec. 18, 1953 because of a urethral stricture. There was an incidental history of repeated episodes of cramping upper abdominal pain, usually accompanied by vomiting. There were no gastrointestinal complaints between these episodes. Nothing of importance was found on physical examination. Laboratory findings were within normal limits. A roentgenologic study of the stomach showed a polypoid lesion of the gastric antrum which could be seen to prolapse through the pylorus into the duodenum (fig. 3). At operation a polyp, measuring approximately 2 cm. in diameter with a long pedicle, was found on the greater curvature 1.5 cm. proximal to the pylorus. The tumor was removed and on histologic examination was found to be a benign adenomatous polyp.

Case 5. C. N. T., a 42 year old white man was admitted to the Kansas City Veterans Administration Hospital on Aug. 6, 1953 because of burning pain in the upper abdomen and

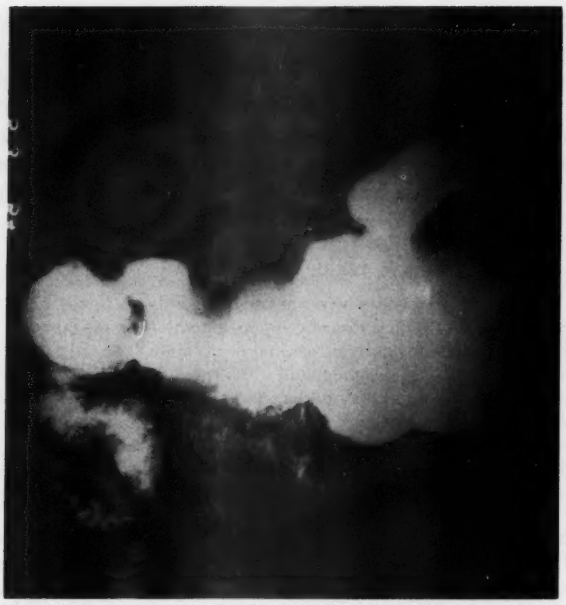

FIG. 1. Polypoid lesion of gastric fundus found to be a leiomyoma following removal (case 2).

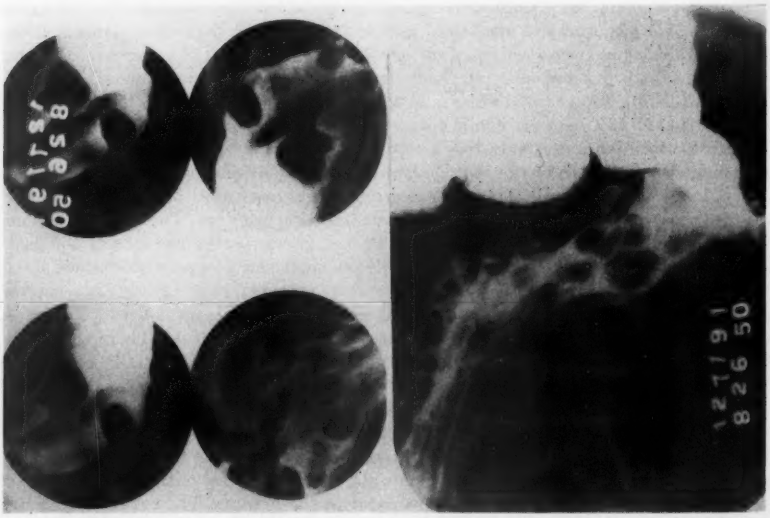

FIG. 2. Multiple polyps in gastric antrum found to be adenomas following partial gastrectomy (case 3).

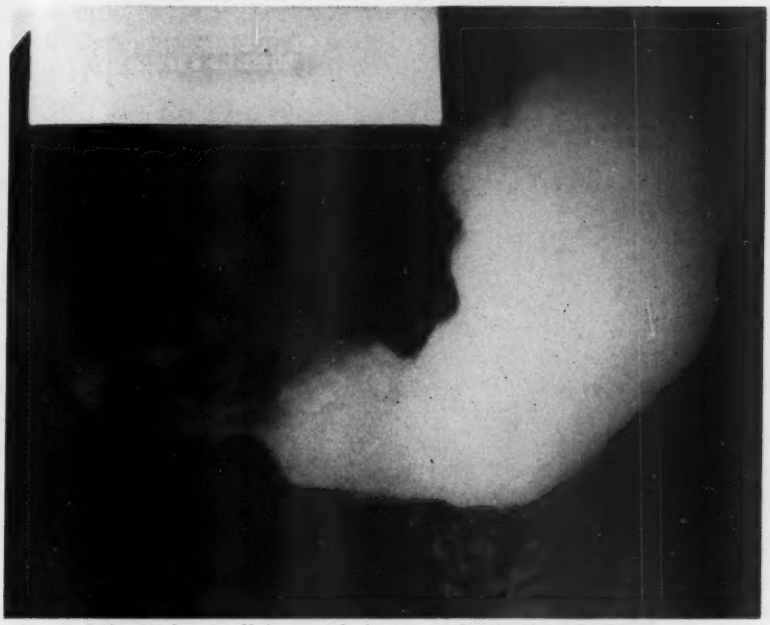

FIG. 3. Polyp on long stalk in prepyloric area prolapsing into duodenum (case 4).

a weight loss of 104 pounds. He had noted vague upper abdominal pain beginning approximately five years before, but had not sought medical treatment until one year prior to admission. He stated that he had had 15 upper gastrointestinal roentgenograms during the year prior to admission and had been told repeatedly that he had a nervous stomach. Physical findings were unimportant, except for evidence of weight loss. Except for a persistent leukocytosis, the laboratory findings were within normal limits. Two gastrointestinal roentgenologic series were reported as normal. By gastroscopy an ulcerated polypoid lesion was seen on the anterior stomach wall just distal to the cardia. A third roentgenogram showed a polypoid filling defect in the fundus of the stomach. A total gastrectomy was done. There were numerous lymph nodes in the area of the celiac vessels. The lesion measured 9 by 10 cm. and on microscopic examination was found to be a reticulum cell lymphosarcoma. The patient was given supplemental deep radiation therapy. He was asymptomatic one year following his operation.

Case 6. W. P., an 81 year old white man was admitted to the Kansas City Veterans Administration Hospital on Oct. 1, 1952 because of upper abdominal pain and anemia. A diagnosis of pernicious anemia had been made in 1946 and at that time a lesion was demonstrated in the prepyloric area of the stomach. On physical examination there was evidence of senility and arteriosclerotic heart disease. Laboratory findings showed a hemoglobin of 8 Gm. The stool examination was strongly positive for occult blood. It was believed that the patient had pernicious anemia as well as chronic blood loss anemia from the gastrointestinal tract. A large polypoid lesion on the greater curvature of the stomach was shown on a roentgenogram (fig. 4). He refused operation and was discharged from the hospital. He was readmitted to the hospital two weeks later because of tarry stools, saying that he would accept operation. At operation a polypoid lesion, measuring 5.5 by 4 cm. with a long pedicle was found on the greater curvature at about the mid portion of the stomach. There were firm nodes in the adjacent gastrocolic ligament. Because of the patient's age, a wedge resec-

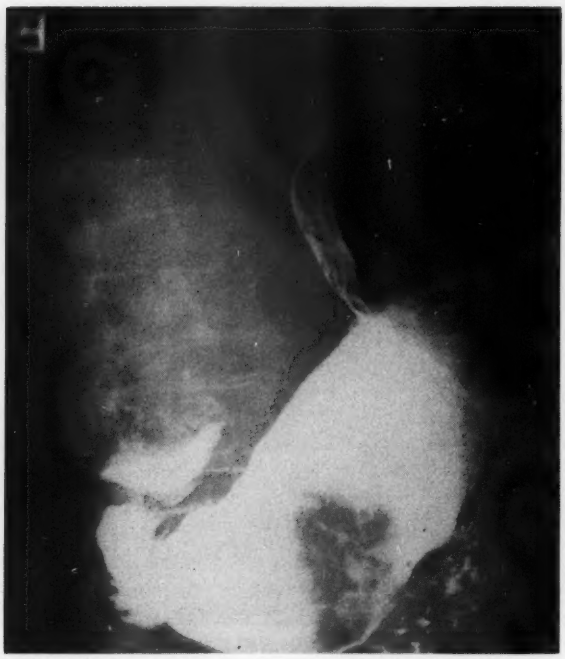

FIG. 4. Polypoid lesion on greater curvature found to be adenocarcinoma following removal. A small adenoma was also found on the lesser curvature (case 6).

tion of the stomach about the polyp was done. There was also a polyp 1 cm. in diameter on the lesser curvature of the stomach which was removed by local excision. A polypoid adenocarcinoma with involvement of adjacent lymph nodes, and a benign adenomatous polyp were reported by the pathologist.

Case 7. R. V. G., a 34 year old white man was admitted to the Wichita Veterans Administration Hospital on Sept. 21, 1951 complaining of chills and fever. There was a previous history of hypertension. During the past month prior to admission there had been anorexia and weight loss of 15 to 20 pounds. There also had been several episodes of tarry stools. The important findings in examination were a blood pressure of 195/90 and a systolic apical heart murmur. The hemoglobin was 5.3 Gm. and the stools were strongly positive for occult blood. A polypoid lesions 4 cm. in diameter in the fundus of the stomach on the posterolateral wall was seen in the roentgenogram. A polypoid lesion in this location also was noted on gastroscopy. Whole blood was given preparatory to surgical exploration; however, the patient suddenly experienced severe upper abdominal pain radiating to the back and began vomiting blood and died before definitive treatment could be instituted.

The autopsy diagnosis was polypoid liposarcoma of the stomach with retroperitoneal metastases. Death was the result of massive retroperitoneal hemorrhage.

CLINICAL FEATURES

Since polypoid growths include a wide variety of lesions, the severity and nature of symptoms is quite variable. Of the 96 cases studied by Thompson and Oyster[23] only one-third of the benign tumors were symptomatic, the remainder

being routine autopsy findings. On the other hand, all of the malignant tumors had produced symptoms leading to clinical diagnoses. These findings are similar to those of Hay who reported 47 per cent of patients with benign adenomas to be asymptomatic while only 1 patient out of 28 with malignancy was asymptomatic.

Except for symptoms of vague epigastric distress, benign polypoid lesions when they do produce symptoms, will do so by bleeding or by causing pyloric obstruction. Bleeding may be sufficient to produce hematemesis (case 1) or tarry stools (case 2) or, more often, will be of a chronic nature producing anemia (case 3). The anemia usually will be the hypochromic type, but in those cases associated with pernicious anemia the picture may be mixed. Most polyps occur in the pyloric antrum and will produce symptoms of intermittent pyloric obstruction when prolapse through the pylorus occurs (case 4). Symptoms manifested by malignant polypoid lesions are likewise nonspecific. Polypoid carcinomas are more prone to bleed than other types of carcinoma and patients commonly present themselves with hematemesis or symptoms of anemia (case 6). On the other hand, bleeding is a less prominent symptom of gastric sarcomas. Weight loss, pain and a palpable mass are the more common early findings (case 5).

It is thus obvious that a correct diagnosis rarely will be made on the basis of clinical findings. Because symptoms, when present, usually are referable to the stomach, a barium study of the upper gastrointestinal tract usually gives the first diagnostic hint. When located in the antrum (and the majority of polyps are located in this area)[1] roentgenologic diagnosis is easy and reliable. However, the difficulty of roentgenologic demonstration of such lesions in the gastric fundus is well illustrated by the patient in case no. 5 who had 15 gastrointestinal roentgenologic studies over a period of one year before the lesion was found. The difficulties in roentgenologic diagnosis have led to a wider recommendation of routine gastroscopy on patients with any upper gastrointestinal complaints.[16] In one series[7] 40 per cent of patients in whom polyps were seen gastroscopically were reported as negative by roentgenogram. It is obvious that a high index of suspicion, coupled with careful roentgenologic and gastroscopic studies, is necessary for an improved diagnostic percentage.

TREATMENT

From the evidence presented it is obvious that all polypoid tumors of the stomach must be considered malignant, or at least premalignant, until histologic proof to the contrary is obtained. This proof is available only following surgical removal of the lesion. The only exception to this rule will be cases in which intercurrent disease or debility makes the risk of surgical intervention greater than the risk of the lesion being or becoming malignant. Since the seventh and eighth decades are the most common age groups in which these lesions are found, there will be some patients in whom the advisability of removing a small polyp which is asymptomatic will be a matter of fine surgical judgement. In our opinion, there is little, if any, place for the so-called *observational management*

of these cases. If the patient's general condition prohibits surgical removal of the polyp, it is unlikely to improve sufficiently to permit surgery if subsequent *observations* reveal changes in the lesion suggestive of malignant degeneration. The extent of operation to be done may not be determined easily. Simple removal of a single pedunculated polyp through gastrotomy, with frozen section examination to ascertain possible malignancy, is recommended. Examination of the stomach with a sterile sigmoidoscope at the time of operation may be useful to determine the presence of other polyps. On the other hand, it already has been pointed out that gastric adenomas often are associated with generalized atrophic gastritis and that carcinomas may develop in a site away from the polyps. These factors favor more extensive removal of the stomach. Therefore, subtotal gastrectomy is the procedure of choice when multiple polyps are present, or in the younger age group.

CONCLUSIONS

A grouping of polypoid gastric lesions is based upon morphologic rather than histologic characteristics and thus may include any type of gastric neoplasm.

Symptoms of these lesions are in no way specific and the clinical diagnosis usually is made by roentgenologic or gastroscopic examination.

Since the determination of cell type, as well as benignancy or malignancy, can be made only by pathologic study, all such lesions should be removed surgically.

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RUPTURE OF THE DUODENUM DUE TO NONPENETRATING ABDOMINAL TRAUMA*

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Abdominal injuries may be divided into two types: (1) percutaneous—resulting from penetrating injuries in which the superficial wound is immediately apparent, emphasizing the possibility of underlying visceral injury, and (2) subcutaneous—resulting from blunt force applied directly or indirectly to the abdominal wall. Injuries to underlying viscera in these patients may be unsuspected and difficult to diagnose.

Nonpenetrating or subcutaneous injuries of the abdomen, reported periodically in the literature, remain a serious problem with an alarming mortality rate. Counseller and McCormack[6] surveyed the literature up to 1935 and found 1313 cases of subcutaneous rupture of the intestine, with a 73.4 per cent mortality rate. There were 113 cases of duodenal rupture, 447 jejunal ruptures, and 412 ileum ruptures. Poer and Woliver[22] added 163 cases to Counseller and McCormack's series from 1935 to 1941, totaling 1476 cases (reported in the literature to this time) of nonpenetrating intestinal injuries, with a decrease in mortality rate to 50.4 per cent in patients operated upon, and an over-all mortality rate of 61.3 per cent. Kelly,[15] in reviewing 157,344 patients admitted to the Albany Hospital over a 15 year period found 74 patients with nonpenetrating abdominal trauma, with a mortality rate of 44 per cent.

Cottrell[5], in 1954, collected 791 cases of nonpenetrating injury to the abdomen, with the following distribution: Kidney—28 per cent; spleen—25 per cent; liver—18 per cent; and the gastrointestinal tract—10 per cent. In a further breakdown of nonpenetrating visceral injuries, we find the incidence of duodenal rupture estimated at 5 per cent to 10 per cent of all subcutaneous gastrointestinal injuries, with a mortality rate between 65 per cent and 95 per cent.[2, 6, 7, 8, 9, 10, 15, 18, 21, 24]

The segments of small intestine most commonly involved are those relatively fixed namely: the duodenum and jejunum near the ligament of Trietz, and the ileum just proximal to the ileocecal valve. Statistics show that 80 per cent of such injuries occur in the terminal ileum and the jejunum just distal to the duodenojejunal flexure and 10 per cent each in the duodenum and colon.[5, 12, 15]

Rupture may occur in three ways: 1. The crushing type in which the viscera is forced against a fixed structure such as the spine or pelvis. 2. A tangential force tearing the bowel and/or mesentery from its attachments. 3. Increased intraluminal pressure due to gas, fluid and intestinal contents trapped between two fixed points. Andrews,[1] using compressed air, showed that a force of 6 to 10 pounds (2.7–4.5 kilograms) will rupture the intestine.

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The clinical findings will vary considerably, depending upon the degree of trauma, the presence or absence of associated injuries, and the general condition of the individual prior to injury. The delayed onset of symptoms has been well reviewed in the literature.[3, 6, 7, 12, 15, 18, 22] In many instances the patient gets up and walks away from the accident, failing to develop clinical symptoms for as long as 12 to 24 hours from the time of the accident. This delay is believed to be due to the gradual escape of intestinal contents before clinical peritonitis develops. The physical signs will, in most instances, reflect the degree of peritonitis.

All too frequently over-reliance is placed on roentgenologic examination of the abdomen in an effort to establish a diagnosis of a perforated hollow viscus. Free air can be demonstrated in 80 per cent of the perforations of the stomach and first portion of the duodenum, and 100 per cent of those with perforation of the colon.[23] However, demonstration of free air following perforation of small intestine occurs in less than 33 per cent of the patients.

Ficarra,[7] reporting 14 cases in nonpenetrating intestinal injury, found only 4 patients demonstrating free air. Jacobson and Carter,[19] reporting 19 cases, found gas under the diaphragm in only 2 of the patients.

These figures are quoted in spite of roentgenological examination made as long as 24 hours after injury. Gas is not visualized normally in the small intestine in adults, and as such the failure to demonstrate free intraperitoneal air in the majority of cases of small intestinal perforations is not surprising. False reliance on the roentgenologic finding of free air in perforation of the small intestine can result in an unnecessary delay, with increased morbidity and mortality rates.

The alarming mortality figures mentioned previously are believed to be due to the failure of early recognition of these lesions, and the frequent occurrence of associated injuries. It has been demonstrated clearly that the mortality rate increases with the delay in diagnosis and operative intervention.[3, 6, 7, 18, 22, 25] Wright and associates[30] reported a mortality rate of 15 per cent of small bowel perforations when detected and repaired within four hours; 44 per cent mortality rate when repaired between four and eight hours, and 70 per cent when repaired after the twelfth hour. Poer and Woliver[22] reported in their series of cases a mortality rate of 35 per cent when the patient was operated upon in less than 12 hours (14 cases), and with a delay of more than 12 hours the mortality rate doubled to 70 per cent (17 cases).

Associated injuries, especially cerebral injuries, may mask the physical findings. Many of these patients are moribund, disoriented and in shock, with multiple injuries taking the focus of attention from the abdomen where potentially fatal, nonpenetrating injury may be present.

In the differential diagnosis, rupture of the diaphragm, injury to the back, thorax, head, abdominal wall, and retroperitoneal structures must be considered. Retroperitoneal perforations have been well reviewed in the literature.[4, 11, 13, 14, 16, 17, 19, 20, 26, 27, 28, 29]

The important consideration is an awareness of the possibility of nonpenetrating injury to the abdominal viscera in all patients presenting themselves

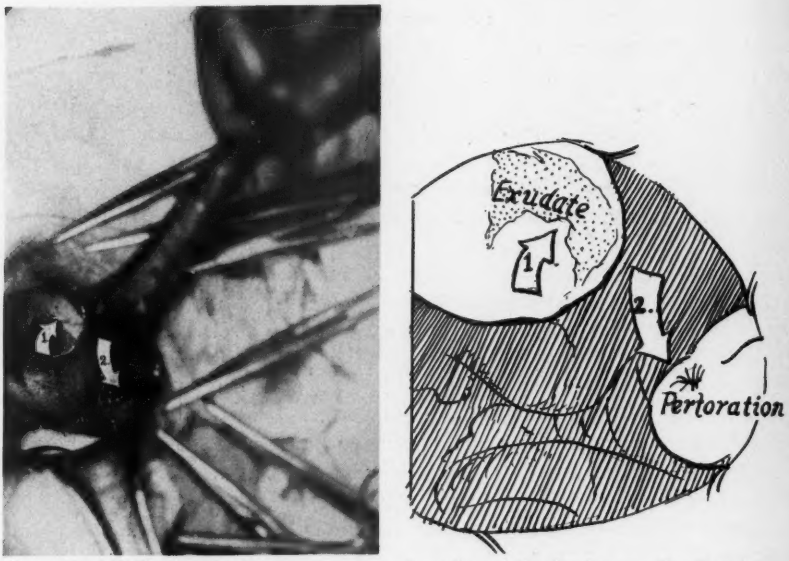

FIG. 1a and b. Demonstrating rupture, second portion of the duodenum. (See line drawing.) Arrow No. 1—demonstrating fibrinous exudate of liver. Arrow No. 2—pointing to perforation of the duodenum.

with a history of direct or indirect trauma to the abdomen. Although the diagnosis of nonpenetrating abdominal injuries may be difficult, signs of intra-abdominal injury will in time be detected with thorough and meticulous clinical observation.

CASE REPORT

N. Y. H. The following case is that of a 2 year old white boy with a subcutaneous perforation of the second portion of the duodenum. This child presented a history of normal development, and was in excellent health until one hour prior to admission to the hospital on Sept. 22, 1952 at which time the following accident occurred:

The mother stated that she had stepped out of the room for a few moments and returned to find the child acutely ill beneath an artificial fireplace, which was lying across the mid-portion of the child's back, with the youngster in the prone position. The fireplace was described as a large, wooden structure. The child did not cry, but remained quiet, pale, apathetic, lethargic, and vomited one time prior to admission.

Physical examination at the time of admission showed a temperature of 37.8 C. rectal, pulse 100, respiration 28, and blood pressure 130/90. The child obviously was acutely ill with grunting respirations. The chest was clear. The abdomen was soft without tenderness, guarding, or change in peristaltic activity. The rectal examination was negative.

Laboratory studies were as follows: Urinalysis—specific gravity 1025; reaction acid. No cells were seen. Hemoglobin was 14 Gm. White blood count showed 17,000 white blood cells per cu. cc. Mature polymorphonuclear leukocytes were 72 per cent, lymphocytes 8 per cent and monocytes 5 per cent. Roentgenologic examination: The skull, chest, lumbar and dorsal spine were negative. Multiple roentgenograms of the abdomen to demonstrate free air, using lateral decubitus, upright and supine films, were negative. The electrocardiogram was normal. Spinal tap, including manometrics and fluid analysis, was normal.

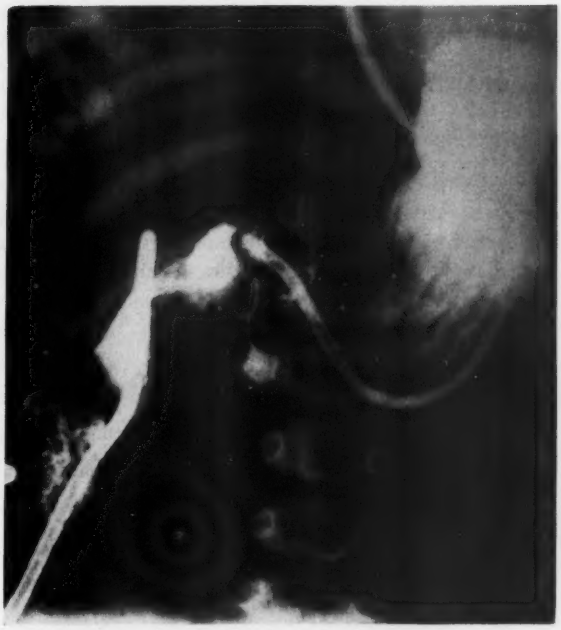

FIG. 2. Fistula of duodenum

The child was placed under constant observation and approximately six hours after admission, for the first time, deep palpation showed moderate guarding and a suggestion of tenderness. The significant finding at this time was the absence of peristalsis. The pulse was 120, respirations 28, blood pressure 130/90, and temperature 38 C. Eight hours after admission to the hospital, on the basis of the development of signs indicating peritonitis, an exploratory laparotomy was done. Endotracheal anesthesia with cyclopropane and ether was used. A left transverse incision was made, without finding any evidence of pathology. However, further examination showed a gush of bile arising from the right lumbar gutter. The incision was extended, and a rupture was found in the anterior portion of the second part of the duodenun just distal to the ampulla of Vater (fig, 1, a and b). There was extensive edema about the porta hepatis with a fibrinous necrotizing bile stained exudate overlying the duodenum, liver, stomach, omentum, and small intestine in this area. The duodenum was mobilized, closed transversely, using continuous atraumatic chromic catgut reinforced with interrupted Lembert No. 0000 silk ligatures. A rubber tube and Penrose drain were placed down to the closure site.

Postoperatively, the youngster was maintained on intragastric suction. Antibiotics were administered, giving 400,000 units of penicillin intramuscularly daily, and intramuscular streptomycin, 250 mg. daily. The patient was maintained on intravenous therapy until the fifth postoperative day when good peristalsis was noted and oral fluids were started. Milk was noted to be coming from the drain site, and a barium swallow (fig. 2) showed evidence of a duodenal fistula, probably arising from the site of closure. The drainage catheter was attached to suction immediately. Electrolyte balance was controlled carefully with intravenous replacement therapy. The drainage remained minimal and the twelfth postoperative day the patient was started on an oral diet which he tolerated well. Sump suction was discontinued and the drainage tube gradually shortened. Gastrointestinal roentgenograms

made 21 days after operation showed no further evidence of fistula. On the sixteenth postoperative day the child had a temperature of 39 C. A blood culture was positive for aerobacterium aerogenes. Intravenous terramycin was administered. The temperature became normal in 48 hours and all antibiotics were discontinued 15 days later. The child, in good condition, was dismissed on Oct. 25, 1952, 34 days after admission. When seen in follow-up, he was in excellent condition, gaining weight, and was free of all complaints.

SUMMARY

This case of a 2 year old white boy, believed to be the youngest patient with a nonpenetrating perforation of the duodenum, reported in the literature, serves to illustrate the difficulties encountered in diagnosis, surgery and management of such lesions. A review of the statistics and the alarming mortality rate found in subcutaneous abdominal injuries is presented.

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GALLSTONE ILEUS*

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Obstruction of the intestinal tract by a gallstone has long been recognized as an occasional complication of gallbladder disease. The incidence reported by various authors varies from 1.3 per cent to 7.8 per cent of all complete small bowel obstructions. Of 76 patients with obstructions reported by Haggstrom and Rousselot[3] an impacted gallstone accounted for 6.5 per cent, placing it as the fourth most common cause of acute intestinal obstruction in their series. McLaughlin and Raines[6] in a review of the literature found that biliary fistulas were present in from .12 per cent to 5 per cent of autopsies studied by various observers. Moskin and Tannenbaum[8] collected from the literature 10 cases of gallstone obstruction occurring more than once in the same individual and added 1 case of their own.

Stones reach the intestinal tract through various routes. Erosion through the wall of the gallbladder can produce adherence of the organ to the duodenum, stomach, or colon with the eventual passage of a stone into the intestinal tract (fig. 1). Regler, Bowman, and Noble[9] found that stones large enough to obstruct the intestine almost always enter by means of true internal biliary fistulas. In one of our patients, however, (case 5), the stone apparently entered the intestine through the ampulla of Vater. Once the intestinal tract is reached, Manfredi[5] has found that a calculus 2 cm. or more in diameter may cause obstruction. The obstruction occurs most commonly in the terminal ileum, but also has been reported in the sigmoid and stomach. The site of obstruction may be in any portion of the small intestine. The vomiting of gallstones has been described by Shapiro and Williford,[10] and others, and occurred in 1 of our patients (case 7). Warner and Swan[12] reported a case of obstruction 2.5 feet proximal to the ileocecal valve by a large stone 2.5 years following cholecystectomy. The authors believed that the calculus had entered the small bowel through the duodenum. The largest stone on record causing intestinal obstruction was 3 by 2¼ by 7″ (7.6 by 5.6 by 17.7 cm.) in size. It was reported by Turner[2] to be impacted in the colon. This stone is now on exhibit in the museum of the Royal College of Surgeons in England.

In spite of the improvement in the diagnosis and treatment of gallbladder disease in general, the mortality rate of this single complication remains extremely high. Although a few small series have been reported with no mortality[6, 10] the fatality of this condition as recorded in the literature varies from 44 per cent to 70 per cent, with an average mortality rate of nearly 50 per cent. There has been little improvement in mortality rate, except for the few series noted above, since the 1890 mortality rate of 44 per cent reported by Courvoiser

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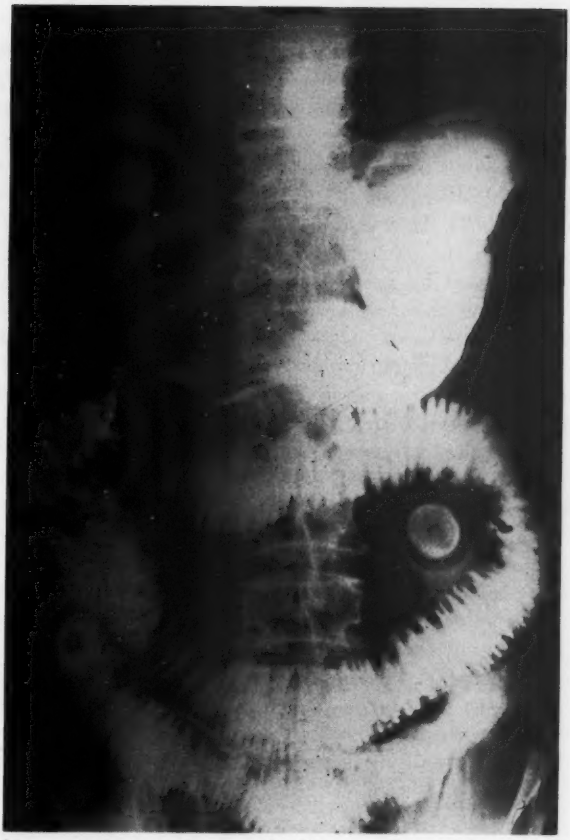

FIG. 1. In this roentgenogram a laminated gallstone is shown obstructing the small intestine. (Courtesy of Dr. William W. Stanbro.)

in 125 cases.[6] Most authors agree that the factors responsible for poor results and a fatal outcome in so many cases of gallstone ileus are the advanced age and obesity of the patients, the presence of masking symptoms, water and salt imbalance, and a lack of awareness of this complication by physicians and surgeons. Murphy is said to have remarked that, "the patient frequently dies, cured of her obstruction."[2]

A review of a large number of cases from the literature, and of our own patients, has convinced us that a fatalistic approach to this problem is not justified. Two remedial factors of the greatest importance in the high mortality rate are errors in diagnosis and delay in treatment. In the hope that by repeatedly calling attention to this condition these factors can be reduced or eliminated, the following cases are presented from the hospital services of the George Washington University School of Medicine and the Medical College of Virginia.

CLINICAL SIGNS AND SYMPTOMS

The symptoms and signs of gallstone ileus are those of acute small intestinal obstruction superimposed, in about 50 per cent of cases,[6, 11] upon those of chronic gallbladder disease. That the complication is more common in females than males is borne out by the fact that 6 of the 7 cases in our series were in women. Although the average age of our patients was 61, that reported by other authors was even higher. In about half the patients symptoms of gallbladder disease have been present before the obstruction occurred. As in other instances of small intestinal obstruction the patient begins to complain of cramping abdominal pain, soon accompanied by nausea and vomiting. Abdominal distention and constipation may or may not be present depending upon the level of the obstruction. Since in many instances the stone becomes lodged high in the jejunum there may be little or no distention and little evidence of small bowel dilatation to be seen in a scout roentgenogram. Recurrent vomiting alone, with varying degrees of upper abdominal pain, may be present, resulting in unwarranted delay in the institution of surgical exploration. By the time these patients reach the hospital they sometimes are in a semicomatose state, complaining of little pain, but demonstrating evidence of severe dehydration, oliguria, and shock with markedly altered electrolyte balance (case 2). In such instances of late obstruction no peristaltic sounds may be audible. If the gallstone lies high in the small bowel, dilated loops of small intestine may not be seen in the roentgenogram and the correct diagnosis of the condition may easily be missed. Of some diagnostic value may be the finding in 5 of the 7 cases of this series of a leukocytosis greater than 14,800. Fortunately in all 5 of our cases in which roentgenograms of the abdomen were taken, evidence of small intestinal obstruction was present. Fever was present in most instances, in 1 case as high as 106 F. Tenderness, although usually noted, was not an outstanding symptom.

TREATMENT AND RESULTS

Although various forms of treatment have been recommended for this condition, most authors agree that early surgical intervention with simple removal of the calculus from the intestinal tract is indicated. Since these patients usually are very ill, any attempt to remove the gallbladder or close the internal fistula at the time of the original operation is contraindicated.[1] The frequent recurrence of gallstone obstruction in the same individual,[7] however, indicates, that when recovery from the first operation is complete, the gallbladder should be removed with closure of the internal biliary fistula at a second procedure. This two stage operation was used in 2 of our patients; the stone was removed from the intestine without further treatment of the fistula in 2; the entire operation was done in one stage in 1; and 2 died without operation. There were three deaths in this series of 7 cases, a mortality rate of 43 per cent. Two patients died without operation due to errors in diagnosis. The remaining death (case 7) occurred in a 56 year old woman during the closure of the internal fistula.

CASE REPORTS

Case 1. A 67 year old white woman was hospitalized on Sept. 13, 1952 with the following history: She was awakened at 1 a.m. on September 12 with vomiting and generalized abdominal pain. The pain was cramp-like in nature and poorly localized. On the day before admission she had passed two large watery stools but had had none since that time. There had been no melena, chills, or fever.

Physical examination revealed an obese, lethargic, elderly white woman. The temperature was 106 F. (taken by rectum); blood pressure 115/80; pulse 90; and respirations 22. The breath sounds were distant throughout the chest. The heart was moderately enlarged to the left, with rhythm regular without murmurs. The abdomen was not distended. There was generalized moderate abdominal tenderness which was poorly localized. Peristalsis was hyperactive on auscultation but did not seem to be of the obstructive type.

Flat and upright roentgenograms of the abdomen showed distention of several loops of small bowel but no fluid levels were present. Hematologic examination showed 15.2 Gm. of hemoglobin and a white blood cell count of 17,300 per cu. mm. with 84 per cent polymorphonuclears. The urine contained 2 plus albumin and 20 to 30 white cells, but no sugar or acetone. The blood urea nitrogen was 37 mg. per cent.

With a tentative diagnosis of severe enteritis a Levin tube was passed and the stomach kept on constant suction. Bile-stained fluid was removed in moderate quantity. On the third hospital day abdominal distention appeared and an increase in the quantity of dark bile-stained fluid was aspirated through the stomach tube. The blood urea nitrogen rose to 53 mg. per cent. Roentgenograms of the abdomen at this time showed definite evidence of obstruction of the jejunum and operation was advised.

Operation: Through a right paramedian incision the abdomen was explored and the small bowel was examined, beginning at the ligament of Treitz. It was found to be moderately distended down to the midileum, at which point a hard mass 3 cm. in diameter was found distal to which the intestine was collapsed. The mass was removed through a longitudinal incision and proved to be a gallstone. The bowel was sutured transversely. Examination of the right upper quadrant of the abdomen revealed an inflammatory mass including the gallbladder in which there were several stones. The common duct was explored, and a T tube was inserted. The gallbladder was then removed and the fistula between it and the duodenum was exposed and closed in layers.

On the first postoperative day moderate right pneumothorax was discovered which subsided after evacuation of air through an underwater tube drainage. After five postoperative days she continued to have fever of 102 F. (taken by rectum) but thereafter the temperature gradually came down to normal. There was a considerable quantity of drainage around the T tube but a cholangiogram showed the tube to be in proper position in the common duct. Convalescence was slow but recovery was complete after removal of the T tube.

Case 2. A 69 year old white woman was hospitalized on Sept. 6, 1953 because of nausea, vomiting, weakness, and lethargy. A known diabetic, she was admitted to the Medical Service and was found to have 4 plus sugar in her urine. She had senile cataracts. She had had a cyst removed from her thyroid three or four years previously.

Physical examination on admission revealed a temperature of 100.2 F.; pulse of 96, grossly irregular; respirations 24; and blood pressure 120/70. She was a moderately obese white woman, very drowsy, but apparently mentally clear. The chest was symmetrical, resonant, and clear. The heart was grossly irregular in rhythm with a rate of 120. There were no murmurs. There was no abdominal tenderness; no masses or viscera were felt. Palpation of the abdomen, however, resulted in a renewed attack of vomiting. An electrocardiogram showed auricular fibrillation, uncontrolled. The blood sugar was 168 mg. per cent. The urine was 4 plus positive for sugar but negative for acetone. The white blood cell count was 17,800 per cu. mm. with 82 per cent polymorphonuclears. The diabetes was controlled with insulin but the vomiting continued. A roentgenogram of the abdomen showed several distended loops

of small intestine and no gas in the large bowel. A 2.5 cm. right midabdominal density, possibly a biliary calculus with gas proximal to it, was seen. A Miller-Abbott tube was passed into the stomach and duodenum and placed on constant suction.

On the following day examination of the abdomen elicited no evidence of pain and there was no rebound tenderness. Peristaltic sounds were high-pitched, consistent with mechanical small bowel obstruction. A diagnosis of small bowel obstruction was made, probably due to an impacted gallstone.

Operation: On September 8, through a right midrectus muscle-splitting incision, the abdomen was opened and a considerable quantity of thin, brownish-colored fluid was aspirated. The small intestine was distended to approximately three times its normal diameter. A gallstone, measuring 6 by 4 by 3 cm., was found a short distance below the ligament of Treitz. This was removed through a longitudinal incision and the bowel was closed transversely. Postoperatively the patient had a satisfactory course. The Miller-Abbott tube was removed on the day after operation. Her cardiac and diabetic status remained well controlled and she left the hospital on the eleventh postoperative day.

Case 3. A 57 year old white man was admitted to the U. S. Veterans hospital on Nov. 5, 1949 complaining of colicky abdominal pain. He stated that he had been well until three weeks prior to admission when he began to notice intermittent episodes of rhythmic upper abdominal discomfort, accompanied by vomiting. The attack for which he was admitted had failed to subside over a period of 24 hours, had become more severe and was accompanied by constant nausea and vomiting. He had had no change of bowel habits, hematemesis or melena, chills, fever, or urinary symptoms.

Physical examination on admission revealed no abnormalities except in the abdomen. There was generalized abdominal tenderness which increased over a 12 hour period of observation and became localized in the right lower quadrant. No masses were palpable. There was no rebound tenderness. Peristaltic sounds were high pitched and hyperactive. Laboratory studies showed a white blood cell count of 9,700 per cu. mm.; red blood cell count of 4,530,000 per cu. mm.; and a hemoglobin of 13.5 Gm. The differential count was within normal limits. Urinalysis showed no abnormalities.

A diagnosis of acute appendicitis was made and operation was done through a right paramedian incision. The appendix was found to be normal, but two large gallstones were found obstructing the first portion of the ileum. The larger of these measured 3.4 cm. in diameter, was roughly round with a facetted surface. The smaller stone measured 2.4 cm. in diameter. The stones were dislodged from the site of obstruction and were removed through a small incision which was closed transversely.

Postoperatively, the patient's course was complicated by the development of a draining sinus in the abdominal wound and phlebothrombosis of the left leg. These gradually subsided. A postoperative roentgenogram of the abdomen showed a constant abnormal gas shadow in the gallbladder area. A barium enema demonstrated that this was not part of the large bowel and the shadow was therefore interpreted as representing an air-filled gallbladder due to a cholecystoduodenal fistula. Stones were not demonstrated within the gallbladder. Since the patient was symptom free it was decided that no further treatment was advisable at this time and he was discharged on Dec. 8, 1949.

Case 4. A 69 year old widow was admitted to the U. S. Vererans hospital on Nov. 19, 1946. The history of her illness began four months previously when she had noticed some distress after eating certain types of restaurant food. On November 9, she began to vomit bile-stained fluid, and complained of right epigastric pain and generalized weakness. She continued to vomit daily until her admission to a private hospital on November 16. Her bowels were constipated except for one episode of diarrhea. During her three days in the private hospital, examination revealed moderately active peristaltic sounds, no jaundice, but moderate abdominal distention with localized tenderness in the epigastrium. A Miller-Abbott tube was placed in the duodenum through which a large amount of dark green, bile-stained fluid drained.

On admission to the U. S. Veterans hospital on November 19, the patient appeared acutely ill, stuporous and an adequate history could not be obtained. Her tongue was dry and

coated, and the abdomen was distended but soft. There was no abdominal rigidity and no masses could be felt on palpation. The blood pressure was 122/74, and the heart sounds were within normal limits. Occasional peristaltic sounds were audible but were diminished in frequency and intensity.

A roentgenogram of the abdomen showed a poorly outlined loop of distended bowel which appeared to be large intestine. No small intestinal dilatation could be seen. There was roentgenographic evidence of some ascites. Laboratory studies showed a white blood cell count of 7,100 per cu. mm. with 75.5 per cent polymorphonuclears; a red blood cell count of 4,550,000 per cu. mm.; and a hemoglobin of 90 per cent. The non-protein nitrogen was 61 mg. per cent; the blood sugar was 169 mg. per cent; and the blood amylase was 215 (normal, 80-180). Urinalysis showed no albumin, casts or sugar.

From the time of admission to the hospital the patient passed almost no urine in spite of the administration of intravenous fluids. A Levin tube was used on gastric suction. The temperature rose from 100 F. to 104 F., pulse from 100 to 120, and the respirations to 24. The patient gradually slipped into a state of deep coma from which she could not be aroused. The non-protein nitrogen rose to 75 mg. per cent. The blood chlorides were 551 mg. per cent; and blood sugar, 201 mg. per cent. She died on November 24, five days after admission.

Postmortem examination: Examination of the lungs showed evidence of bronchopneumonia bilaterally. The abdomen was moderately distended. The gallbladder was small, thick-walled and did not contain stones. It was closely adherent to the first part of the duodenum on its anterior surface, and a smooth, round opening was present between the gallbladder and the duodenum 2.5 cm. in diameter. Two and one-half feet below the ligament of Treitz a large cylinderical shaped gallstone, 4 cm. in length, was obstructing the bowel. The remaining small bowel was essentially normal without distention.

Case 5. A 59 year old housewife was admitted to George Washington University Hospital on April 11, 1938 because of abdominal pain, nausea, vomiting, and constipation of five days' duration. She had had no bowel movement since the onset of her present illness five days previously and had had no prior attacks of pain or vomiting. She had no chills, fever or jaundice. Except for some persistent peripheral edema of long standing and noticeable shortness of breath on exertion, her past history had not been remarkable.

Physical examination on admission showed a temperature of 98 F.; a pulse of 110; and a blood pressure of 155/100. There was some enlargement of the cardiac dullness extending to the left of the midclavicular line. The abdomen was large and obese with marked tenderness throughout. There was no rigidity and no abdominal masses were palpable. There was no rebound tenderness and no costovertebral tenderness. On admission there was a white blood cell count of 20,750 per cu. mm. with a normal differential count. The blood chlorides were 304 mg. per cent; the CO_2 combining power 33 mg. per cent; and blood urea nitrogen 80 mg. per cent. On April 13, 1938 the non-protein nitrogen was 125 mg. per cent and serum chlorides 439 mg. per cent. Urinalysis was within normal limits except for 1 plus albumin and a few white blood cells and red blood cells.

The temperature rapidly rose from 98 F. on admission, April 11, to 104 F. on April 15. The pulse remained 100 throughout. She became irrational and soon slipped into a deep coma from which she could not be aroused. She was treated by gastric suction, intravenous fluids, blood transfusion and supportive therapy, but her condition rapidly deteriorated and she died on April 15, four days after admission.

Postmortem examination was made on April 15. The duodenum was very firmly attached to a mass in the region of the gallbladder and was covered with omentum. On opening the duodenum the ampulla of Vater and the common bile duct were found greatly dilated. The jejunum was greatly distended by gas and was somewhat cyanotic in color. At the junction of the jejunum and ileum a gallstone was found obstructing the intestine which measured 3 by 5 cm. in diameter. The gallbladder walls were greatly thickened and the gallbladder was firmly attached to the liver. No stones were present in it. The cystic duct was dilated so that a finger could be passed easily into the common duct and into the duodenum through it.

Case 6. A 52 year old white woman was admitted to George Washington University

Hospital on July 27, 1953 complaining of cramping generalized abdominal pain with nausea and vomiting of one week's duration. For several years she was known to have a large gallstone which was visible in a roentgenogram of the abdomen, and on several occasions had had attacks of right upper quadrant pain referred to the shoulder and infrascapular region accompanied by vomiting.

One week before admission she began to notice generalized abdominal pain which rapidly became very intense and she began to vomit bile-stained fluid profusely. Her bowel movements became liquid and she had had no formed movements for one week. She took a large dose of milk of magnesia on the night before admission without result, and on admission had passed no flatus for 24 hours.

The temperature was 37 F., and pulse was 98. She was obviously in acute pain. She vomited almost constantly and showed evidence of dehydration. The abdomen was obese; there was slight hyperresonance but no localized tenderness was found. She had recurring attacks of abdominal pain during the examination and at these times high-pitched bowel sounds were heard synchronous with the pain. The urinalysis was normal. The white blood cell count was 17,300 per cu. mm. with 84 per cent polymorphonuclears. The blood sugar was 119 mg. per cent. The hematocrit was 48 cc. per cent. A roentgenogram of the abdomen showed a single loop of dilated jejunum with relatively little gas in the large bowel. Mechanical small bowel obstruction was suggested and an area of calcification was seen at the lower margin of the liver suggesting the possibility of a gallstone.

Operation: Through a left paramedian incision the jejunum was found to be reddened, congested, and moderately distended. At a point near the lower end of the jejunum the bowel was found to be obstructed by a large gallstone measuring 8.3 cm. in diameter. The stone was removed and the incision in the bowel was closed transversely with two rows of sutures. Exploration of the gallbladder area showed a small, shrunken, acutely inflamed gallbladder containing a small stone. A fistulous tract was demonstrated between the gallbladder and the duodenum. No further surgery was done and the wound was closed.

The postoperative course was essentially uneventful and the patient was discharged on August 5. She returned to the hospital on September 3, for removal of the gallbladder and closure of the duodenal fistula.

Operation: On September 24, through a right subcostal incision, the abdomen was opened in the usual manner. Two stones were palpable in the gallbladder but none in the common bile duct. A fistulous tract was found extending from the gallbladder to the second portion of the duodenum. The gallbladder was removed and the fistula between the gallbladder and duodenum was transected and the duodenal end closed. Two ducts were found entering the common duct from the gallbladder, a double cystic duct. The common duct was explored and no stones were found. A T tube was placed in the common bile duct and the wound was closed with drainage.

Postoperatively the patient drained a rather large quantity of bile through the common duct tube and also through the drain for a period of three weeks. A cholangiogram demonstrated that the ampulla of Vater was patent and no stones were seen within the duct. The tube was then removed and the wound healed satisfactorily. She was discharged on September 17, and her postoperative course thereafter was satisfactory.

Case 7. A 56 year old Negro woman was admitted to Gallinger Municipal Hospital on May 24, 1950 with a history of nausea and vomiting of 24 hours duration. Three gallstones were found in the vomitus. On the day before admission, during the afternoon, after eating some corned beef, she suddenly developed intermittent epigastric pain localized in the center of the abdomen and radiating to the lower abdomen. Her bowels had not moved for 48 hours but she continued to pass gas by her rectum.

She was a thin, Negro woman showing evidence of acute illness. The abdomen was soft and nontender throughout except for slight tenderness in the epigastrium. There was no rigidity. The gallbladder was not palpable nor were the liver and spleen. Urinalysis was noncontributory except for the presence of many white cells in a centrifuged specimen. The white blood cell count was 6,150 per cu. mm.; the red blood cell count was 3,030,000 per cu.

mm.; and hemoglobin was 61 per cent. A roentgenogram of the abdomen showed gaseous distention of several loops of small bowel and a small quantity of gas in the colon. There was an irregular area of calcification to the left of the first lumbar vertebra, thought possibly due to a calcified lymph gland. With a diagnosis of partial small bowel obstruction a Cantor tube was passed into the small intestine. In spite of this, she continued to complain of abdominal pain and the output of the Cantor tube continued to be profuse.

Operation: On May 28, four days after admission, the abdomen was explored. It contained a large quantity of straw-colored fluid. Twelve inches above the ileocecal valve a cylindrical gallstone measuring 3 cm. in diameter was found in the lumen of the ileum. The intestine above this point was distended. Through a small incision the stone was removed and the incision was closed transversely with two layers of sutures. The gallbladder was hard and indurated and appeared to be filled with numerous small calculi. Because of the condition of the patient further operation seemed contraindicated.

Postoperatively a low grade wound infection developed which gradually healed, and she was discharged on June 30, her incision well healed.

On July 6 she was readmitted to the hospital for further investigation of her gallbladder and closure of a possible cholecystoduodenal fistula. On admission she complained of abdominal pain and was moderately distended. A small intestinal tube was passed with immediate relief and after removal of the tube she developed an excellent appetite, regained her strength, and was up and about the ward freely. Except for serum chlorides of 439 mg. per cent and a total protein of 5.67 mg. per cent, laboratory studies were not remarkable.

Operation: Through a right rectus incision the peritoneal cavity was opened. A large number of adhesions were found binding the small bowel to the anterior abdominal wall. These were carefully freed and the gallbladder finally located. A fistulous tract about 1 cm. in diameter was found extending into the duodenum. The duodenum was closed and the gallbladder was removed. At this point in the operation the patient's blood pressure fell to 78/58 but returned to normal with the aid of vasoconstrictors. The common duct was then explored which was found to be greatly enlarged but patent. It was drained with a T tube. At the end of the procedure the patient's condition was poor, the pressure having fallen to 70/40 and the pulse having risen to 120. She was kept in the operating room receiving a blood transfusion, positive pressure oxygen, and digitalis, but all measures were without avail and she died one hour after the procedure.

DISCUSSION

It is evident from the cases presented and from those reported by other authors that the principal cause of the high mortality rate associated with intestinal obstruction due to gallstones is failure or delay in making the correct diagnosis. No qualified surgeon would hesitate to do an immediate laparotomy if the diagnosis of gallstone ileus could be established. In only 2 of these cases was the correct preoperative diagnosis made, and the average length of time from the onset of symptoms to laparotomy or death was four and one-half days. This tendency to delay is confirmed by other authors.[4] The site of obstruction in this condition is often high in the small bowel where loss of electrolytes produces such a profound effect, particularly in patients of the older age group who are usually affected by this condition. Factors which lead to errors in diagnosis are the frequent absence of gaseous distention typifying small bowel obstruction on a roentgenogram due to the fact that the obstruction may be so high in the small bowel that there is less intestinal gas to be visualized; and profound changes in the electrolyte balance producing azotemia, dehydration, and even semicoma, with diminishing symptoms and signs leading the surgeon

TABLE 1

Case summary—gallstone ileus

Case No.	Age	Sex	Site of Obstruction	Previous Symptoms	Duration of Attack (days)	Admission Findings	Treatment	Result
1	67	F	Mid-ileum	None	6	Pain, vomiting, diarrhea, temp. 106 F., Wbc 17,300	Removal of stone; cholecystectomy; Choledochostomy	Cured
2	69	F	12 inches below ligament of Treitz	None	5	Vomiting, tenderness, no pain, Wbc 17,100	Removal of stone	Cured
3	57	M	Upper ileum 2 stones	Recurrent pain 3 weeks	1	Pain, vomiting, tenderness, Wbc 9,700	Removal of stone	Cured
4	69	F	24 inches below ligament of Treitz	4 months distress postprandial	8	Vomiting, slight pain, Wbc 14,800, tenderness, T. 100 F.–104 F.	No operation	Died
5	59	F	Junction jejunoileum	None	4	Severe pain, vomiting, tenderness, Wbc 20,750	No operation	Died
6	52	F	Lower jejunum	Pain, vomiting 1 year	3	Vomiting, pain, diarrhea, obesity, Wbc 17,300	Removal of stone; cholecystectomy; choledochostomy later	Cured
7	56	F	Lower ileum	None	5	Vomited, gallstone pain, tenderness, Wbc 6,150	Removal of stone; cholecystectomy; choledochostomy later	Died

to suspect a medical cause for the patient's clinical picture. In addition, an impression still exists that many cases of intestinal obstruction can be treated safely by intestinal intubation and restoration of fluid balance until the diagnosis becomes clear. Gallstone ileus, however, is an example of the type of obstruction which, although not commonly producing gangrene of the intestine wall, nevertheless results in a fatality if treated by conservative means alone.

A symptom complex which should suggest intestinal obstruction due to a gallstone is that of an elderly female patient who has had no previous operation but has had attacks of upper abdominal pain or intolerance to fat foods, and who develops cramping pain with persistent vomiting. On examination there is moderate tenderness, rhythmic pain and peristaltic rushes. With these symptoms the condition must be considered acute high intestinal obstruction and treated as such. Other diagnostic aids, including roentgenograms, blood counts and blood chemistry, are of value. Evidence of small bowel obstruction by roentgenogram was present in 5 of the 7 cases presented above. Elevation of the white blood count above 14,000 per cu. mm was present in 5 of the cases. All complained of vomiting. One had vomited gallstones. All but 1 complained of cramping abdominal pain. Three had had a definite past history suggesting gallbladder disease.

The treatment of this condition is that of small intestinal obstruction, namely, early operation with removal of the gallstone from the intestine and simple transverse closure of the intestine. The patients are often extremely ill. The operative procedure should be limited to the relief of the obstruction alone. It may be possible to examine the gallbladder during the emergency procedure and to determine whether or not more stones are present in it or in the intestinal tract. This is especially necessary if a facetted stone has been causing the obstruction. When the patient has recovered from the operation, re-exploration should be done with removal of the gallbladder and closure of the cholecystoduodenal fistula. One must be careful to detect anomalies of the bile duct since in 1 of the cases presented (case 6) two cystic ducts were present. Any attempt to remove the gallbladder at the time of the original operation may prove disastrous.

SUMMARY

Seven cases of intestinal obstruction due to gallstones have been presented, with a mortality rate of 43 per cent. In 2, no operation was done; in 2, only the obstructing stone was removed; in 2, the obstructing stone and later the gallbladder were removed in separate stages; and in 1, the obstruction was relieved and the gallbladder removed in the same procedure.

The most important factor in the high mortality rate occurring in gallstone ileus is failure or delay in the making of a correct diagnosis and in the institution of surgical intervention.

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ELEPHANTIASIS CHIRURGICA: CAUSE AND PREVENTION

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INTRODUCTION

Lymphedema of the arm following radical mastectomy has defied explanation for many years.[2] The most popular concepts regarding its cause are concerned with lymphatic obstruction. It now is apparent that lymphatic obstruction is not the primary cause of lymphedema. The experiments of DeCamp and associates,[1] have demonstrated the causal relationship between valvular incompetence, high sustained venous pressures, and lymphedema of the leg.

From our experiments, we believe that the indurated upper extremity following radical mastectomy is due to increased venous pressure in the arm, resulting from impairment of venous drainage. Our studies indicate that trauma to the axillary vein during a complete radical mastectomy initiates varying degrees of changes within the vein, varying from a narrowing or partial thrombosis to occlusion and complete thrombosis. The elevated venous pressures are secondary to the interference with venous return, and the elevation of pressure is correlated with the extent of venous blockage. Increased venous pressures generally are temporary following a radical mastectomy and usually subside as the thrombophlebitis is resolved in a period of two to six months. It is during the phase of venous insufficiency, characterized by elevation of venous pressures, that treatment is mandatory, otherwise the edema will become a permanent feature in spite of recanalization of the vein and a corresponding reduction in venous pressure of the arm. Kinmouth and Taylor[3] recently have demonstrated dilatation and failure of lymphatic vessels in cases of congenital edema of the legs. We suspect that similar developments are found in elephantiasis chirurgica late in the disease, but as a secondary effect of the venous incompetence. Since lymphedema once established is a permanent feature, persistent swelling must be explained on the basis of accumulation of interstitial fluid in the subcutaneous space which defies all therapy.

We believe that the only treatment for lymphedema of the arm is prevention. The best therapy for thrombophlebitis is elastic wrapping for control of the swelling until the elevated pressures have subsided. Anticoagulants were tried in this experimental study and discarded as dangerous. In a previous communication[4] we have recommended the use of a routine elastoplast pressure bandage to the upper extremity for a period of two months following radical mastectomy. Such an elastic compression offers the best means of preventing a permanently swollen arm. None of our patients who wore the bandage properly demonstrated more

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than 1 inch increase in circumference of the arm. Not all patients subjected to radical operation will show restitution of the veins to normal within a period of two months, and in many of these, an indurated arm may develop subsequent to removal of the compression bandage. Most arms will show varying degrees of swelling early in the postoperative phase in spite of the elastic wrapping, and it is imperative that the application of the bandage be snug enough to eliminate this swelling. Properly applied, the bandage is tolerated well and does not interfere with motion of the shoulder, or of the joints of the arm, and is welcomed by the patient who has a thorough understanding of its purpose.

MATERIAL AND METHOD

In proof of our concept that venous insufficiency produces elephantiasis chirurgica, experimental studies were made on a series of over 100 consecutive cases of cancer of the breast seen at the University of Oklahoma Hospitals, in an attempt to ascertain the venous changes in the axillary vein, coincident with, and as a result of, radical mastectomy. Serial examinations were made, beginning before operation; at the time of the first dressing—four to seven days postoperative—and at intervals of one to several months thereafter. Patients whose veins were slow to recanalize required the longest period of investigation. All operations in the series, done by the resident surgical staff, had a complete dissection of the axilla, which included intra-adventitial stripping of the axillary vein from its midpoint to at least 3 cm. lateral to the subscapular vein. In many, a metal clip was fastened about the stump of the ligated subscapular vein to better identify the region in subsequent venograms.

To interpret the various physiologic changes that occur after mastectomy, we have relied on venous pressure determinations and venograms in order to follow and correlate the degree of venous embarrassment of the axilla. Measurements of the circumference of the arm on the side operated upon have been correlated to demonstrate the degree of swelling with the foregoing.

The technic of venography, previously described, is briefly; rapid injection of Diodrast into one of the veins of the dorsum of the hand; preferably the same vein is used throughout the entire study. Coincident with the injection of the Diodrast into a vein of the hand, the patient is instructed to execute a Valsalva maneuver, which facilitates filling of the veins of the arm and neck, the extent of which is dependent to some degree upon the unpredictable cooperation of the patient.

In order to correlate pressure changes with the venograms, manometric determinations of the venous pressures, utilizing the same vein in the hand were done on the day of venography. Venous pressures were recorded at rest and during exercise. Contrary to findings in the leg, venous pressures in the arm are increased with exercise.[6, 7] The more venous blockage that is noted, the greater the disparity between active and resting pressures. There is a wide variation of pressure elevation following mastectomy, which is compatible with the degree of injury and blockage of the veins of the axilla. The circumference of the arm was measured at serial intervals comparable to the venogram, and venous pressure studies were made in order to correlate the degree of swelling with changes in the axillary

vein and elevation of venous pressures. The circumference of the upper extremity was measured at three levels: high next to the axilla; low arm, just above the elbow; and forearm, just below the elbow. From practical experience we have learned that the low arm measurement above the elbow is the most sensitive and reflects best the tendency toward any postoperative swelling.

A study of many preoperative venograms indicates that there are two major systems of venous drainage of the arm. The principal drainage is by the axillary vein which is a continuation of the brachial vein. A secondary system, the cephalic, is an auxiliary system, comparable to the saphenous vein of the leg (fig. 1), which assists in returning blood from the upper extremity. The cephalic may be called upon as the alternate route for return of blood when the axillary vein is compromised. In preoperative venograms the cephalic normally carries only a small quantity of the dye, or frequently none at all. With interference of the axillary vein it may become dilated to assist in returning blood or it may become the sole route of venous return from the arm. The interpretation of venograms is a complex task due to normal variation of venous structures

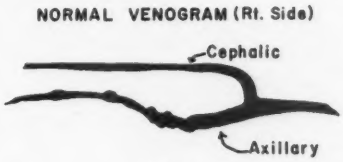

FIG. 1

and the occurrence of venae comitantes which may be made of as many as three venous channels in parallel. The anatomic arrangement of the veins is so designed that utilization of the cephalic vein or venae comitantes modifies postoperative edema in those patients in whom the operative trauma has damaged the axillary vein to the extent of occlusion. There is a complex system of valves in three or four areas of the axillary vein, the most constant appearing at the junction of the subscapular within the axillary, and at the junction of the cephalic with the axillary. In this group of patients, the cephalic was called upon to assist the damaged axillary, but not to totally replace it, so that increases in venous pressure were minimal. This type then should have the least rise in venous pressures and swelling of the arm as shown in table 1.

It again should be pointed out that the narrowing was maximal in the region of the subscapular junction with the axillary vein. We believe that the change is in the nature of a valvulitis with thrombus formation, particularly on the caudal side where the surgical trauma is maximal.[5] We do not believe that venospasm would produce such constant deformities and over such prolonged periods as the venograms were repeated.

In order to illustrate this type of venous grouping, the case of P. G. is shown (fig. 2). This patient demonstrated only slight narrowing of the axillary vein with slight increase of venous pressure (from 11 to 12 cm. water on exercise) and no tendency to swelling. There actually was evidence of atrophy of the arm

TABLE 1

Category A (17 cases)

	Edema	Venous Pressures Recorded During Exercise		
		Initial	Highest	Final
		mm.	mm.	mm.
Type I venogram (7 cases)				
1. P. G.	−¾″	110	120	110
2. L. P.	−¼″	110	120	120
3. E. C.	0	120	195	120
4. P. G.	0	—	—	—
5. A. K.	+¼″	110	160	120
6. A. S.*	+¾″	110	160	150
7. V. R.	+1″	140	150	150
Average edema	+¼″	Aver.: 120	Aver.: 150	
		Average increase: 30 mm. H_2O.		
Type II venogram (5 cases)				
1. L. F.	+¼″	160	280	150
2. M. O.	+¾″	120	170	140
3. W. T.	+¾″	100	180	130
4. L. D.	+¾″	140	240	160
5. F. D.	+1¼″	150	200	170
Average edema	+¾″	Aver.: 130	Aver.: 210	
		Average increase: 80 mm. H_2O.		
Type III venogram (4 cases)				
1. M. H.	+1″	100	240	150
2. M. T.†	+1½″	130	170	120
3. M. W.†	+2″	110	200	120
4. M. S.	—	—	—	—
Average edema	+1½″	Aver.: 120	Aver.: 210	
		Average increase: 90 mm. H_2O.		
Type IV venogram (1 case)				
1. O. W.‡	0	140	280	180
		Increase: 140 mm.		

* Wore no bandage at any time.

† Removed bandage at five weeks.

‡ Wore Ace bandage for several months after routine elastoplast.

— Insufficient data.

postoperatively, which was not an unusual finding in the absence of swelling and probably is related to limitation of use (see table 1, cases shown with smaller arms postoperatively). The very minimal pressure increase in this patient fell to normal within two months; there was no evidence of increased pressures at rest (chart 1).

Table 1 lists all patients who demonstrated type I venogram changes; they

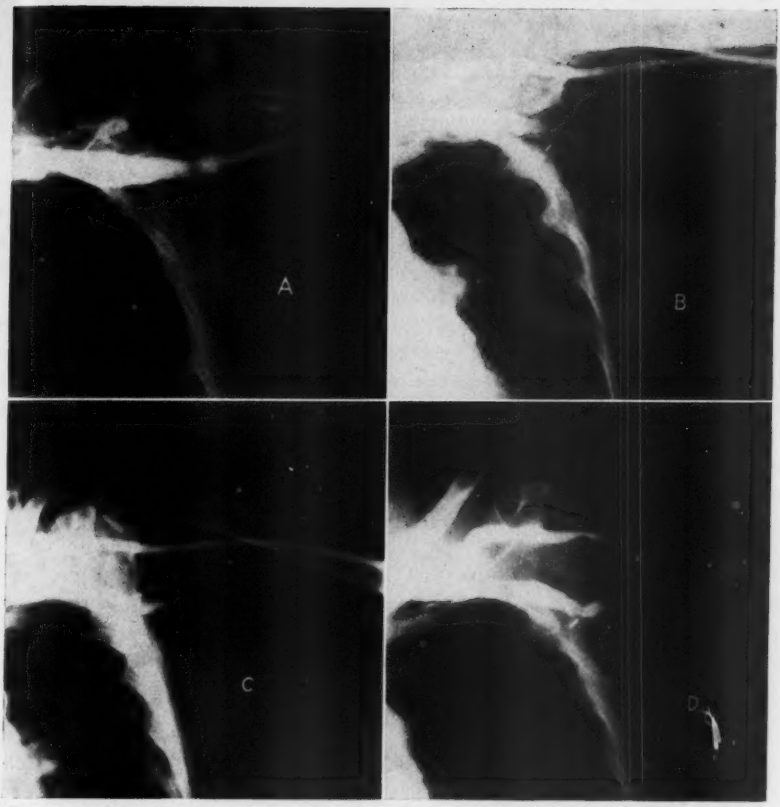

FIG. 2. P. G., white woman aged 56. Type I venogram. A—Preoperative film showing good filling of the axillary vein. No cephalic demonstrated at this time. B—Six days postoperative. Axillary vein very narrow laterally. Cephalic vein now prominent and carrying most of the dye. C—Two months postoperative. Axillary vein still narrowed and carrying only a small amount of dye. Cephalic continues to carry most of the blood. D—Seven months postoperative. Axillary vein almost restored to normal caliber. The cephalic vein continues to carry some of the dye. Valve at junction of subscapular with axillary vein appears functional.

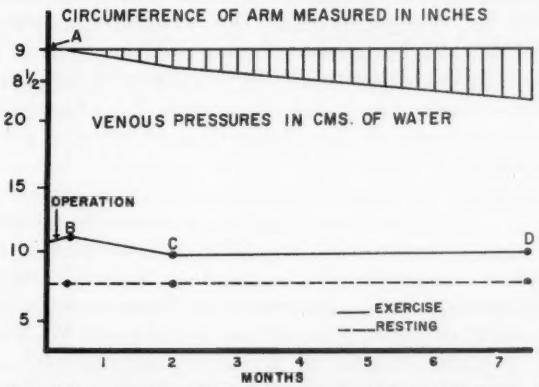

CHART 1. P. G., white woman aged 56. Pressures recorded at A, B, C, & D determined at time venograms, A, B, C, & D of figure 2. Very slight rise in venous pressure is correlated with slight distortion of the veins of the axilla postoperatively. This curve is typical of the group of type I venogram. Decrease in circumference of the arm is thought to be due to disuse.

are 7 in number. Note that only 2 patients in this group showed any appreciable swelling of the arm and both evidenced prolonged elevation of pressure (cases 6 and 7). Type I venogram complex thus consists of that group of 7 patients who demonstrated only slight narrowing of the axillary vein with good function of the cephalic vein postoperatively, and with minimal pressure changes, which, in turn, is reflected in least tendency to swelling. The average increase in active pressures following operation averaged 30 mm. of water.

The final conclusions of our experimental study were gleaned from evidence compiled from 33 patients whom we were able to follow in greatest detail. Further classifying the 33 patients, category A represents 17 patients who had carcinoma of the breast and were treated by radical operation alone. Category B represents 7 patients who had carcinoma of the breast with metastases to the axilla who were treated by radical operation and postoperative radiation therapy. Category C represents 9 patients who had incurable cancer, according to the criteria of Haagensen and Stout, who were treated by palliative radiation therapy.

Investigative data from the 17 patients treated by operation alone illustrates: (1) the nature of venous obstruction; (2) the increased venous pressures and; (3) swelling of the arm incident to mastectomy. Venous changes in this group of patients varied from very slight damage to complete occlusion of the major veins. According to the degree of distortion of the veins within the axilla types I II, III, and IV venograms are defined. The most profound changes occur in type IV, the most minor in type I.

ANALYSIS OF RESULTS

Type I Venogram. The first type of change to be recognized is in the 7 patients who had a functioning cephalic vein and who developed only slight narrowing of the axillary vein postoperatively. This is the minimal change to see in the axilla. In none of our patients did the vein look undisturbed or unnarrowed following a radical mastectomy; or stated conversely, radical operation resulted in distortion to some degree in every one of the axillary veins which were studied. Two of the 7 patients who showed type I change in the axilla failed to visualize the cephalic vein in the preoperative roentgenograms. But, as axillary narrowing developed postoperatively, the cephalic functioned as an auxiliary system. The average swelling demonstrated in patients with type I venogram was 0.25 inch. Any elevation of pressure usually was quickly readjusted. This group probably would do well with no wrapping of the arm. Forty-two per cent of all patients studied had type I venogram.

Type II Venogram. Venogram type II represents those patients who had a functional cephalic vein and a normal axillary vein prior to operation, but who developed a complete blockage of the axillary as evidenced by failure to fill in the postoperative venograms. The 5 patients in the group (table 1, type II) evidenced more elevation in the postoperative venous pressures and this was reflected in a tendency to more marked swelling of the arm. W. T. is an example of this group and demonstrated both a good cephalic and axillary vein in the preoperative venogram (fig. 3). The axillary was absent completely on the postoperative films and remained so for four months. (See venogram, figure 3,D

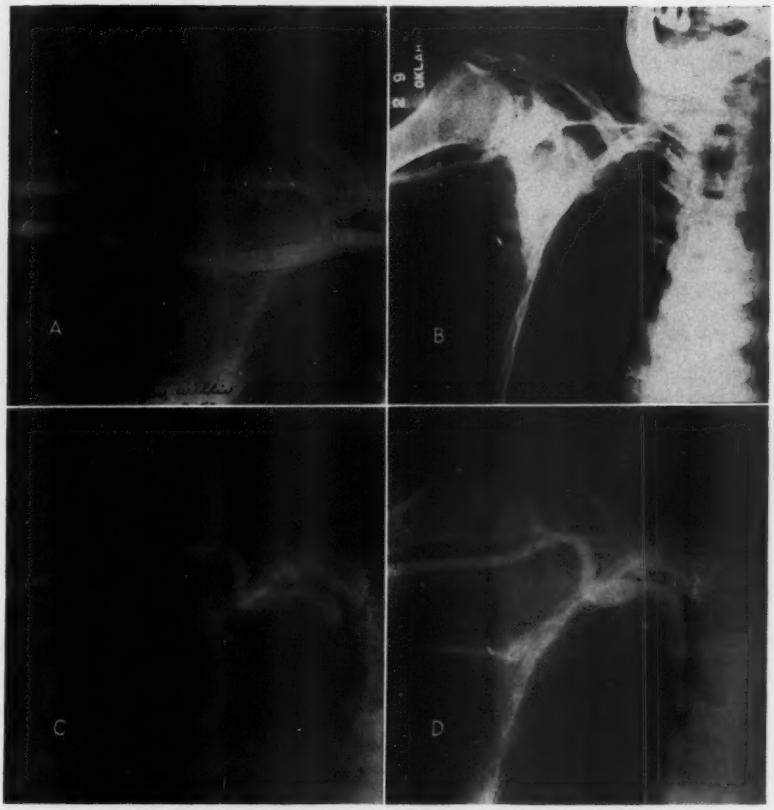

FIG. 3. W. T., white woman aged 65. Type II venogram. A—Preoperative film showing good filling of the axillary and cephalic veins. Numerous valves shown within the axillary vein and at junction of major tributaries. B—Six days postoperative. Axillary vein blocked lateral to junction with cephalic vein. Cephalic vein carrying all the dye through the lateral axilla. Note that the medial extent of the axillary vein is patent. Metal clip is on stump of the subscapular vein. C—Six weeks postoperative. Axillary fails to fill lateral to junction with cephalic vein. Cephalic is more dilated. Only one channel carrying the dye (cephalic). D—Four months postoperative. Cephalic vein still carrying most of the dye. Axillary vein is recanalized but small in caliber.

axillary beginning to recanalize at four months.) The pressure rose 80 mm. of water, from 100 to 180 mm. and remained somewhat elevated at the last reading six months postoperative. She developed 0.75 inch swelling of the arm in spite of well maintained compression of the arm. Venograms demonstrated that the cephalic vein had assumed the major role of returning blood from the arm. Although it had functioned well in this capacity, since it is not functionally adequate as compared to the axillary vein, elevated pressures and swelling resulted (chart 2). A review of type II cases shows the more profound pressure rises, with more sustained elevation of pressures. Four of 5 patients had 0.75 inch swelling or more.

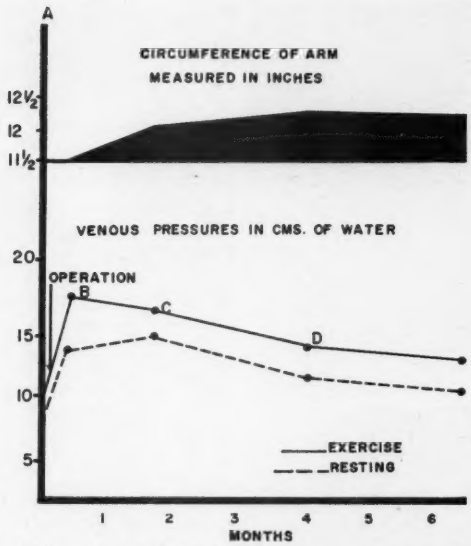

CHART 2. W. T., white woman aged 65. Pressures recorded at A, B, C, & D determined at time venograms, A, B, C, & D of figure 3. Compare elevation of pressures with extent of venous blockage. This curve is typical of those patients with moderate embarrassment of venous return. Greatest elevation of pressure during exercise amounted to 80 mm. After six and one-half months, the pressure is not quite back to normal. Swelling of arm was slow to develop, but was persistent.

The type II group averaged an 80 mm. rise in active pressure after mastectomy. The swelling in this group averaged 0.75 inch in spite of the fact that all 5 wore their bandages faithfully. Thirty per cent of all patients studied had type II venogram.

Type III Venogram. Type III venogram is that group which was characterized by the absence of a cephalic vein throughout all sequences. It seems unlikely that 4 of the 17 patients would have a congenital absence of the cephalic. We believe, on the other hand, that it is not functioning in draining the hand or forearm in this group, but probably is present anatomically. All attempts to show the cephalic in this group by injecting the lateral aspect of the anticubital space have been fruitless. All 4 of these patients had excellent function of the axillary in preoperative films and operation resulted in narrowing of the axillary without complete thrombosis. Type III patients then have no cephalic vein, and only narrowing of the axillary vein. Thrombosis never was complete in the axillary vein in this group, although venae comitantes, when present, were used to good advantage. There were 4 patients who fell into this type venogram. (See table 1, type III.)

As an illustrative case of type III that of M. W. is reproduced (fig. 4). The postoperative roentgenograms showed no cephalic, narrowing of the axillary, and a prominent vena comitans. The main channel of the axillary vein was constricted for five months. The active venous pressure rose 90 mm., from 110 to

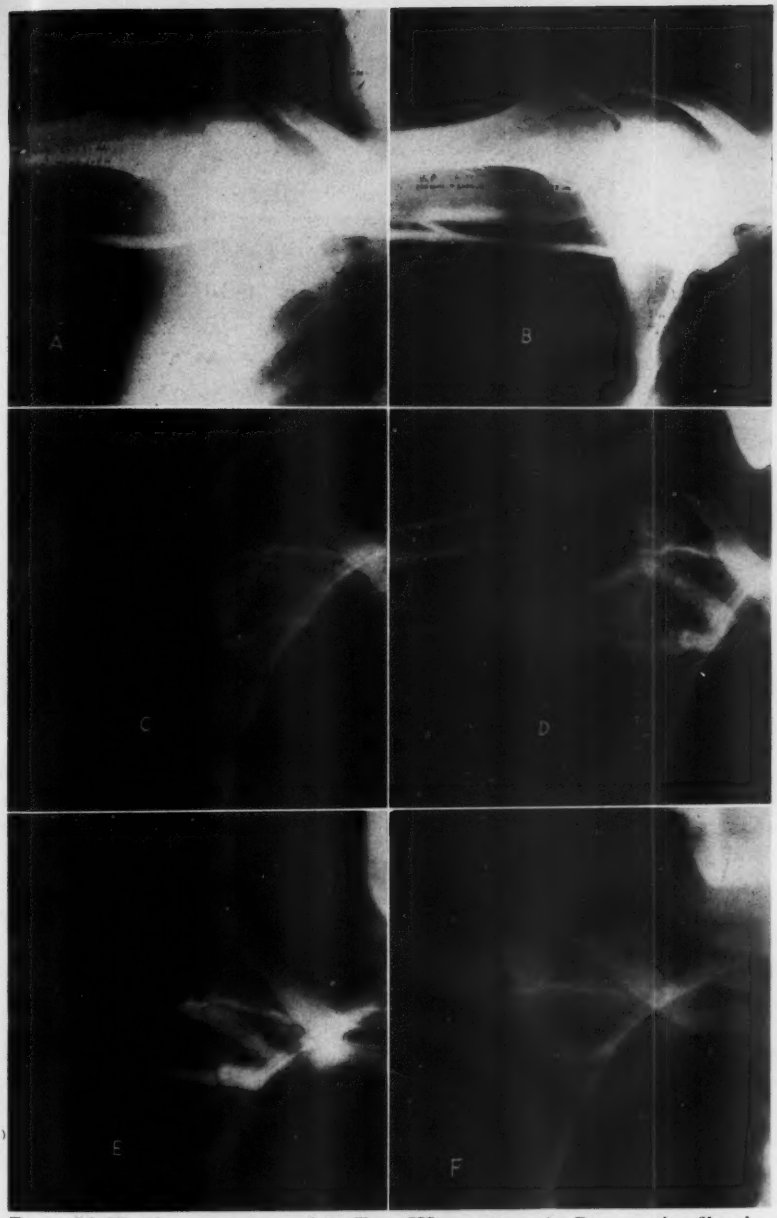

FIG. 4. M. W., Negro woman aged 60. Type III venogram. A—Preoperative film showing large, normal appearing axillary vein with even caliber throughout. No filling of the cephalic vein. Note failure of cephalic to fill throughout this series. B—Eighteen days postoperative. Axillary vein reduced in diameter. Note the appearance of venae comitantes superior to the narrowed axillary vein. (No cephalic vein.) C—Two and one-half months postoperative. Axillary vein still reduced in diameter. Venae comitantes appear more dilated. (No cephalic.) D—Five and one-half months postoperative. Essentially as before. E—Eight months postoperative. Axillary vein increased in caliber and now carrying more of the dye. Venae comitantes dilated medially. F—twenty-one months postoperative. Note that film is similar to the preoperative film A. Almost perfect restoration of the normal return of dye. Axillary vein now carrying all the injection and the venae comitantes have disappeared. The caliber of the restored axillary vein is about as shown in preoperative views. (No cephalic.)

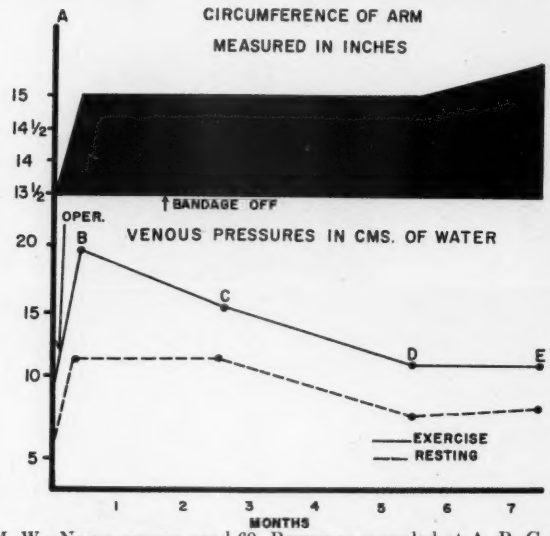

CHART 3. M. W., Negro woman aged 60. Pressures recorded at A, B, C, D, & E determined at time venograms, A, B, C, D, & E of figure 4. Absence of cephalic and narrowing of the axillary veins has resulted in more elevation of pressures. Greatest elevation of pressure occurred immediately postoperatively and amounted to 90 mm.

200 mm. of water. After five months the pressure fell satisfactorily, but the patient developed 2 inches of permanent swelling of the arm. We attribute this swelling to poor cooperation and refusal of the patient to wear a bandage (chart 3).

Type III patients average 1.5 inches swelling of the arm. Two of the 4 patients who developed the greatest swelling wore the bandage only five weeks. Average elevation of pressure following mastectomy was 90 mm. of water, or three times the average rise in type I patients. Twenty per cent of all patients studied had type III venograms.

Type IV Venogram. Type IV venogram is demonstrated by the 1 patient who, postoperatively developed a block to both the cephalic and axillary veins. The preoperative films had shown good function of both veins. O. W., a patient with severe diabetes whose skin flaps sloughed, and in whom we were able to see the thrombosed segments of both the cephalic and axillary veins. The thrombotic process was fairly gradual in onset and one was able to see in subsequent venograms the development of collateral circulation about the axilla (see venogram figure 5). She developed very high pressures during the course of the illness, the increase being 140 mm., from 140 to 280 mm. of water. During the period of greatly increased pressures, the arm swelled 1.75 inches, but with prolonged compression the arm returned to preoperative circumference. We attribute this to prolonged bandaging with Ace elastic after the routine two months of elastoplast. An additional element that we had not anticipated was the evaluation of edema in these patients who terminally lost a great deal of weight as this one did (chart 4).

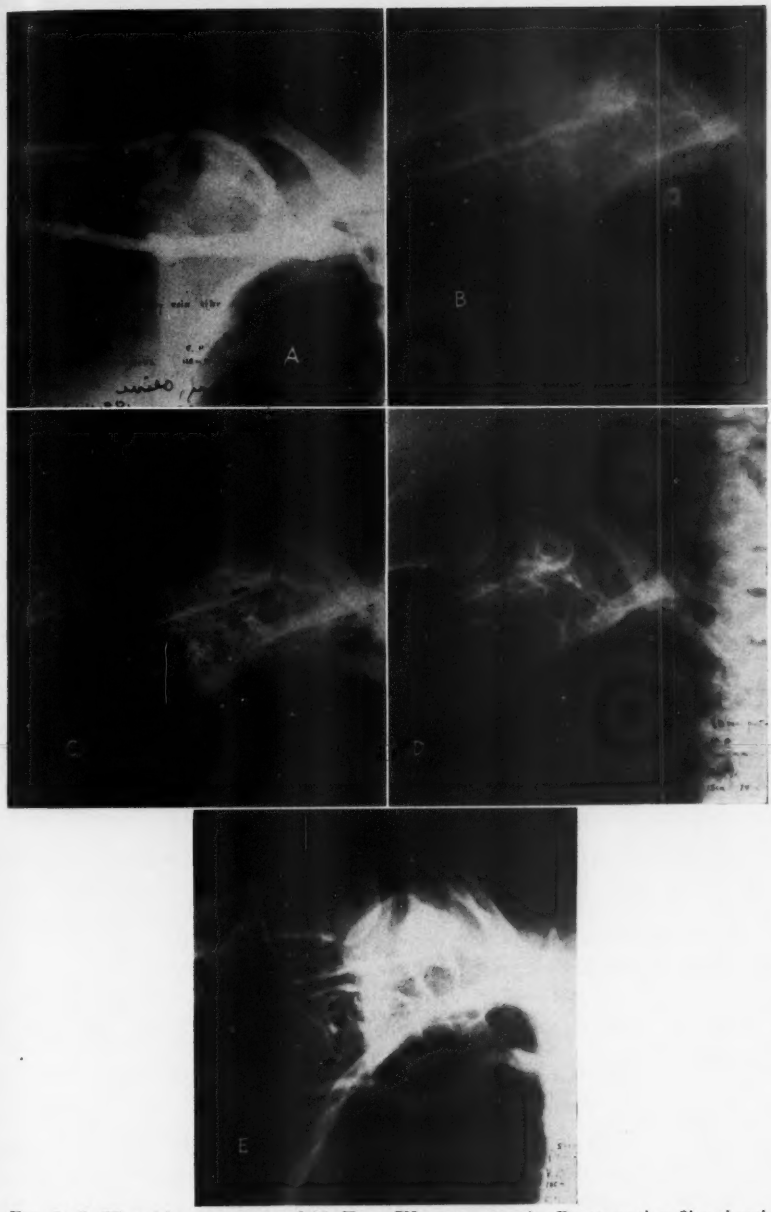

FIG. 5. O. W., white woman aged 68. Type IV venogram. A—Preoperative film showing normal appearing axilla with cephalic and axillary veins both well filled. Note the prominent valves within the axillary vein. B—Ten days postoperative. Cephalic vein blocked. Axillary vein blocked but fills to lateral border of the axilla. Venae comitantes carrying most of the dye to midaxilla but with no direct shunt to the axillary medially. C—One month postoperative. Cephalic blocked. Axillary vein blocked. Venae comitantes now two in number with collateral veins to the medial part of the axillary vein or to the subclavian. Many collaterals are shown about the shoulder. D—Three months postoperative. Axillary vein has recanalized but is reduced in caliber throughout the axilla. Venae comitantes have direct communication with the medial end of the axillary vein. E—Six and one-half months postoperative. Axillary vein has recanalized but is reduced in caliber. No valves visible within its lumen. Venae comitantes are prominent and have network of anastomoses about the medial axilla. (The cephalic had sloughed.)

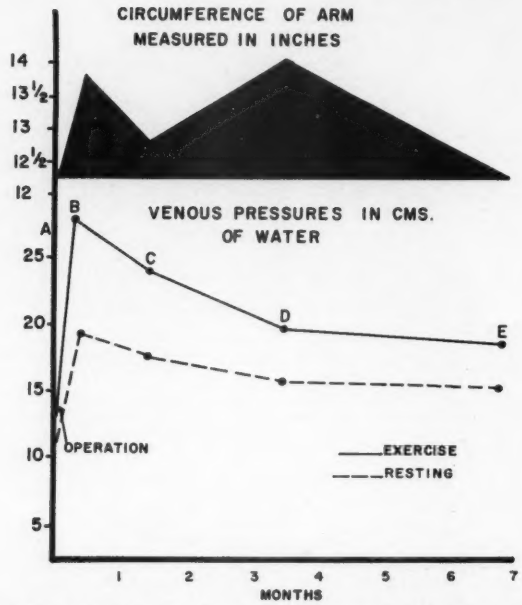

CHART 4. O. W., white woman, aged 68. Pressures recorded at A, B, C, D, & E determined at time venograms, A, B, C, D, & E of figure 5. Thrombosis and sloughing of the cephalic in addition to thrombosis of the axillary vein resulted in very high postoperative pressure. Swelling at one time reached 1¾ inch but continued elastic bandage eliminated this. This patient lost a great part of her weight terminally which may have accounted in part for the absence of swelling of the arm.

Conclusions drawn from 1 patient are not reliable, but in the group of patients treated with operation plus radiation therapy (category B to be reported later) there is a second patient, who had operative ligation of the cephalic due to the proximity of metastases. She then developed a complete axillary thrombosis with the result that her venous pressures increased from 90 to 310 mm. of water. The rise of 220 mm. was the greatest we encountered and she subsequently developed 3 inches of swelling. Only 8 per cent of all patients studied had type IV venograms.

DISCUSSION

It must be concluded then that the degree of interference within the veins affects the venous return from the arm which is reflected by a rise in venous pressure, and the pressure in turn causes swelling of the arm. The degree of venous distortion has been divided into four groups on the basis of characteristic features found on the venograms (fig. 6). It is to be emphasized that the four types of venous complex are determined by the appearance of the postoperative venograms alone. The types were useful in that all cases could be categorized and they assisted in interpreting the many and varied data.

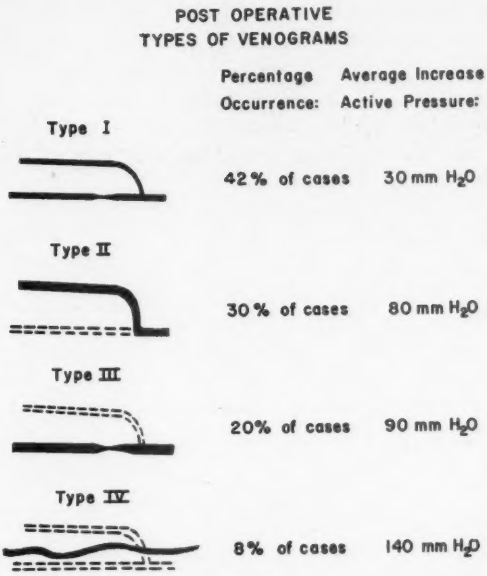

FIG. 6

The degree of venous distortion is variable. The entire process is transitory and eventually returns to normal within a period of two to six months; only the swelling seems to be lasting. One patient, however, required two years for the axillary to return to normal. The degree of distortion probably depends on the extent of surgical trauma inflicted upon the vein plus an unknown tendency for intravenous clotting on the part of the patient. We obtained routine prothrombin time, whole and diluted 1:12.5 on all of our patients, but were unable to draw any conclusions. The individual anatomic or functional variation also exerts an influence on the degree of pressure rise and swelling by denying the patient at times an alternate route of venous return (absence of cephalic vein), or failure to equip her with venae comitantes to assist venous return when the axillary and cephalic veins are damaged.

It would seem that we have gone to great lengths to try to make a very complicated explanation for a simple phenomenon—postoperative edema of the arm. When this work was started no one had any idea that the subject would be so complicated. It may be that a great deal has been read into the venograms, but the other data has substantiated such a grouping of venogram types. It seems to the authors that no other explanation fits the experimental findings.

It is impractical to subject all patients to postoperative venous pressure determinations or to venography. The many suggestions for eliminating swelling in mastectomy patients seem unsound. Since traumatic thrombophlebitis is the basis for swelling, it is practical to subject all patients to elastic compression of the arm until the process has subsided.

Treatment of established edema of the arm is disappointing. Prevention of swelling is the goal that must be attained. As can be seen in this group, not all patients subjected to mastectomy will have enough axillary embarrassment to develop edema. About 42 per cent of our patients had type I venous changes and probably needed no bandage. However, none of these patients was the worse for having worn the elastic. Treeves has stated that about 60 per cent of patients can be expected to develop appreciable swelling of the arm. This agrees well with our findings, which offers a reasonable explanation of why the 60 per cent do swell. In our group, types II, III, and IV made up 58 per cent of the series, and, on the basis of these experimental findings, they could be expected to swell.

Finally, it must be admitted that elastic compression is not a perfect way to eliminate lymphedema, as inspection of the data in table 1 will show. It will reduce swelling to a minimum, however. The use of such substances as fibrinolysin eventually may replace all other therapy, but for the present compression bandage seems to be the method of choice. The bandage should be applied as early as possible postoperatively and maintained until the venous pressure elevation has subsided.

CONCLUSIONS

A report on 17 patients who were studied prior to and at intervals after radical mastectomy is submitted.

Varying degrees of damage to the veins of the axilla results from a complete radical mastectomy.

The pattern of venous obstruction after mastectomy is unpredictable, but can be classified into any of four types by examination with venography.

Venous embarrassment, denoted by reduction in caliber of the vein or occlusion of the vein, is associated with elevation of venous pressures.

Edema of the arm results from the rise in venous pressure, and the degree of swelling corresponds with the degree of pressure elevation.

The traumatic thrombophlebitis and elevated venous pressures are temporary and are reversible processes.

Swelling of the arm, on the contrary, becomes a permanent feature unless controlled when the venous pressures are high.

Routine application of an elastoplast compression bandage to the arm is recommended as the best means to escape elephantiasis chirurgica.

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JEJUNOGASTRIC INTUSSUSCEPTION: AN UNUSUAL COMPLICATION OF GASTROENTEROSTOMY

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In the 73 years which have elapsed since the first gastroenterostomy was done there have been about 50 reported cases of acute retrograde intussusception of the jejunum through the stoma. The first instance of this unusual complication was recorded by Steber in Germany in 1917, and most of the subsequently reported cases have appeared in the European literature. Only 8 instances of this condition have been reported in this country up to the present time.

Bettman and Baldwin[3] contributed a comprehensive review of the literature on this subject in 1933 when they reported a case in which 120 cm. of gangrenous jejunum was found in the stomach at operation. Gottesman[6] reported an acute case of jejunogastric intussusception in 1936, and similar cases have been reported by Ducey and McNamara,[5] Shackman,[11] Lawson and Whitener,[8] and Bansmer.[2] McNamara[9] has recorded a case of retrograde jejunogastric intussusception through a subtotal gastrectomy stoma. Lavida, Haynes and Debakey[7] have recently described a similar case which occurred eight years following subtotal resection for carcinoma of the stomach. There have been 4 other instances of jejunogastric intussusception following subtotal gastrectomy.

In addition to the acute and often fatal type of jejunogastric intussusception a much milder condition of chronic, intermittent intussusception has been reported a number of times in the literature. This seems to be a rather peculiar and distinct entity, and such patients do not come to surgery unless the attack fails to subside promptly. The diagnosis is made roentgenologically. Details regarding this form of intermittent intussusception have been reported by Sibley[12] and by Butler, Wooley and Burton.[4] Aleman[1] also discusses this type in a comprehensive review of 70 cases of jejunogastric intussusception following all forms of gastric operations.

The *mechanism* by which this condition is produced consists of a strong antiperistaltic action in the efferent loop of jejunum. Except in the case of the chronic intermittent type, which may be the result of an abnormally large stoma and which has its onset shortly after gastroenterostomy, the pathology is essentially the same in every case. On opening the abdomen at operation or necropsy a large, distended stomach is found with a coiled, sausage-shaped mass of invaginated efferent loop protruding from the stoma into the lumen. Where the stoma is large and the intussusception is of short duration the invaginated portion of bowel may be reduced easily by traction and often is viable. In cases of longer duration, especially in those patients with tight stomas, the bowel is gangrenous and the mucosa presents a necrotic, hemorrhagic appearance. According to

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Bettman and Baldwin the size of the stoma in reported cases varied from 2 to 5 cm. in diameter and the length of invaginated small bowel varied from 5 cm. to 2 M. The age at which this condition developed has been reported to be from 21 to 75 years, and it has appeared from 6 days to 16 years following gastroenterostomy.

The *etiology* of this complication is obscure. Oselladore[10] has produced experimentally retrograde gastro-gastric invagination in dogs by instilling a weak solution of silver nitrate into the stomach. He postulates that edema in the anastomosis will cause active gastric peristalsis with rapid emptying, and this peristalsis may draw the mucosa of the small intestine into the stoma, thus initiating the invagination. Lawson and Whitener suggest that antiperistaltic action may be induced by hyperacidity of the gastric secretions, and they list as possible accessory factors, (1) the relaxation of the gastric wall, (2) an unusually large stoma, and (3) any sudden increase in intraabdominal pressure. It has been reported 26 times following posterior gastrojejunostomy and 6 times following anterior gastrojejunostomy which may suggest an etiologic relationship with the location of the stoma.

The *clinical picture* is rather characteristic. The onset is typically sudden with acute severe epigastric pain, nausea, and vomiting. The pain is intermittent and colicky in character and tends to increase in frequency and severity as the condition progresses. Vomiting usually is present from the onset and is of an intermittent, frequently projectile type. The vomitus usually consists of clear gastric fluid during the first few hours, becoming darker as bleeding begins from the necrotic mucosa of the prolapsed bowel. It rarely has the coffee ground appearance seen in bleeding ulcer and it may be of a dark red color suggestive of port wine. Physical findings are moderate, diffuse upper abdominal tenderness in the early case, or marked abdominal tenderness and boardlike rigidity in the late case. A palpable epigastric mass has been reported in 17 instances. This latter finding usually is considered to be almost pathognomonic of intussusception, although in at least 1 patient it has led to an erroneous diagnosis of gastric malignancy. Fever usually is absent, and in long-standing cases shock, dehydration and anemia are prominent features.

The *laboratory findings* are of little aid in the diagnosis. Leukocytosis usually is absent or minimal. Anemia is present in proportion to the severity of the hemorrhage, or the hematocrit may be elevated in the presence of shock. Roentgenograms of the abdomen may show a soft tissue mass in the left upper quadrant of the abdomen, and erect films may be of considerable value in ruling out an acute perforation of the peptic ulcer. Fluoroscopic barium studies are diagnostic in the mild, chronic intermittent type of intussusception. In the acute form, especially where there is hemorrhage and shock, such studies usually cannot be made.

Because of the rarity of this complication, a correct *preoperative diagnosis* has been made in only 3 instances in the cases reported. Usually high intestinal obstruction is the chief clinical diagnosis, with bleeding from a stomal ulcer and acute perforation being strongly favored in the later more severe cases. Early

diagnosis is most important as it has been shown that the mortality rate in patients operated upon less than 48 hours after the onset of symptoms was only 10.5 per cent. In those patients operated upon over 48 hours after the onset the mortality rate was 53.8 per cent. Patients not operated upon invariably have died.

Some type of *surgical treatment* was undertaken in 33 patients of the reported cases. The operative procedure has varied with the severity of the condition found on exploration. In the milder cases, where traction alone reduced the intussusception and the bowel appeared viable, simple fixation of the efferent loop has given good results. Occasionally enteroenterostomy may be done on such patients. In the more severe forms, when the stomach is opened to reduce the intussusception, a subtotal gastric resection has been done. The type of anastomosis depends upon whether or not the invaginated bowel must be resected. In those patients in whom the bowel was found to be viable, Polya and Billroth II anastomoses have given good results. The few cases reported in which the bowel was gangrenous and required extensive resection have terminated fatally.

REPORT OF A CASE

On Aug. 9, 1952, a 69 year old white man was admitted to the hospital with a history of severe, intermittent upper abdominal pain of 18 hours duration. The pain was sudden in onset, of a cramping, colicky character and was associated with severe, projectile-type vomiting which tended to recur about every hour. Four hours before admission, the vomitus, previously clear, had become blood-tinged, and at the time of admission it was of dark, burgundy color. There was a past history of a chronic duodenal ulcer treated by gastroenterostomy 11 years previously. He had been essentially asymptomatic as regards gastrointestinal disturbances since then. About three weeks before the onset of the present illness he developed a severe endophthalmitis with progressive loss of vision of the left eye despite intensive therapy with broad-spectrum antibiotics and cortisone. At the time his present trouble began he was scheduled to have an enucleation of the left eye because of symptoms suggestive of sympathetic ophthalmia.

On admission the patient appeared to be both acutely and chronically ill. He was somewhat emaciated and severely dehydrated. He had a temperature of 99.2 F., a pulse of 180, a respiration of 20, and a blood pressure of 110/60. Except for the ophthalmic condition the physical examination revealed only moderate, diffuse upper abdominal tenderness, slight muscle spasm confined to the upper abdomen, and a suggestion of an ill-defined mass in the midepigastrium.

Laboratory data obtained on admission showed a hemoglobin of 8.2 Gm., red blood cells, 3,100,000 per cu. mm., white blood cells, 8,900 per cu. mm. and a negative urinalysis. Roentgenograms taken in flat and erect positions were essentially normal.

The patient was given plasma and intravenous fluid and electrolytes immediately after admission pending typing and cross-matching for blood transfusion. On the basis of a clinical diagnosis of bleeding stomal ulcer he was given vigorous supportive treatment in anticipation of possible surgical intervention. During the ensuing 10 hours he received a total of 1500 cc. of whole blood, but despite this he continued to manifest signs of mild shock. Forceful vomiting of the dark red material continued regularly despite gastric suction, and it was deemed advisable to do a laparotomy in order to secure hemostasis.

At operation a greatly distended, darkly discolored stomach was found which was of rather firm, doughy consistency. After mobilizing the stomach along the greater curvature and freeing numerous adhesions, the posterior gastroenterostomy was visualized, and it was apparent that the efferent loop of jejunum was intussuscepting into the stoma. Traction on the efferent bowel was ineffective in reducing the invagination. The stomach was opened

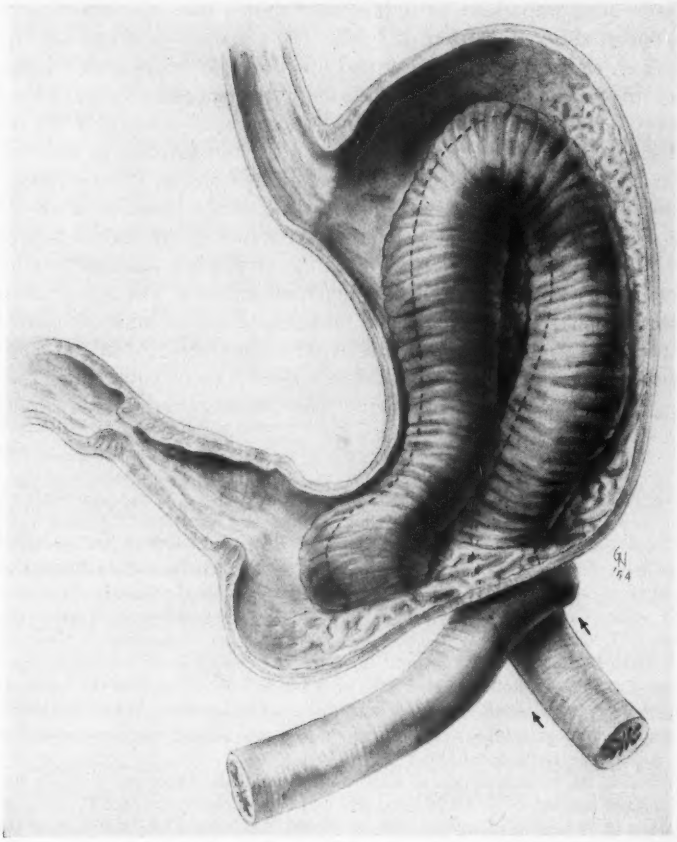

FIG. 1. The above sketch shows the condition found at operation. The mechanism by which the efferent loop of jejunum is invaginated through the stoma of the posterior gastroenterostomy is illustrated. In this instance the edematous, hemorrhagic condition of the invaginated bowel precluded reduction by traction on the efferent loop, and resection was required. A palpable mass in the left upper quadrant in a gastroenterostomized patient with symptoms of high intestinal obstruction is practically pathognomonic of intussusception.

and a large, sausage-shaped mass of inverted jejunum was seen coiled in the dilated stomach. The invaginated bowel was purplish-red in color with a necrotic mucosal surface which was bleeding from innumerable small sloughs.

The duodenum, which had been sectioned previously just distal to the pylorus, was closed and buried in the usual manner and the afferent and efferent loops of jejunum were cut between clamps. The proximal portion of the greatly distended stomach was divided to remove about $\frac{4}{5}$ of it with the inverted jejunal contents. An end to end anastomosis next was made connecting the proximal and distal ends of the jejunum. Because of the extreme dilatation of the stomach it was necessary to employ the Hofmeister modification to complete the Polya type of anastomosis. The resected jejunum measured 38 in. (76.5 cm.) in length.

Continuous gastric suction, which was instituted prior to operation, was continued for

five days postoperatively during which time the patient was supported daily by whole blood transfusions, parenteral fluids and electrolytes, and intravenous dextrose and Amigen. He appeared to be making an uneventful recovery with peristalsis resumed on the second postoperative day and small fluid feedings tolerated on the fifth postoperative day. Sutures were removed on the ninth day. On the tenth day, during an attack of coughing, he eviscerated. Secondary closure was made with through and through sutures. A persistent pancreatic fistula developed. This fistula was presumed to have resulted from manipulation incident to the hastily done gastric resection. It was a source of great annoyance to the patient, who became discouraged and was a severe nutritional problem. It was hoped that his condition might be improved sufficiently to re-explore him in an effort to correct the pancreatic fistula. During the fifth postoperative week he developed signs of circulatory collapse. The electrocardiograph indicated extensive myocardial damage. He died two days after the circulatory collapse.

SUMMARY

The literature regarding jejunogastric intussusception following gastroenterostomy is reviewed and the pathology, etiology and clinical features of this unusual complication are discussed. A heretofore unpublished case is presented, making a total of 9 such cases now reported from this country. Despite the infrequency with which this condition has been reported during the 73 years that gastroenterostomy has been practiced in the management of peptic ulcer it should be strongly considered in any patient with a gastroenterostomy who presents the following picture: intermittent epigastric pain of sudden onset; nausea and vomiting of a forceful type with subsequent hematemesis; and a palpable mass in the epigastrium. Early diagnosis and prompt surgical intervention can greatly reduce the mortality rate in this dangerous complication.

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USE OF FREEZE DRIED HUMAN FASCIA LATA IN THE REPAIR OF INCISIONAL HERNIAS

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Since Gallie[3] in 1921 reported on the use of autogenous fascia for the repair of hernias, many favorable reports[1, 4, 5, 6, 7, 8] have been recorded in the literature concerning its use, particularly in large incisional hernias. The advantages of autogenous fascia used in strips as a suture are; that it causes no tissue reaction, will retain its strength for many months or years in the host tissues and, because of its ribbon-like shape, will not cut through tissues. Its popularity, however, probably has been somewhat limited because of the necessity of an additional incision in the thigh to obtain the fascia. Most surgeons would prefer to use such suture materials as are more readily available rather than go to the inconvenience of obtaining fascia lata from the thigh of the patient.

In July 1954, we began to investigate the use of homologous human fascia preserved by the procedure of freeze-drying or lyophilization. Human fascia lata was obtained from the thighs of suitable subjects at autopsy, cleaned of fat and areolar tissue, divided into strips 1.5 cm. in width, and attached to Gallie needles with no. 36 stainless steel wire according to Gallie's technic. The fascial strips with needles attached then were wrapped with gauze in small bundles, each containing 2, 4, 6, or 8 sutures. They then were placed in jars of normal saline solution containing penicillin and streptomycin and stored at 4 C. in a refrigerator for 24 to 48 hours. Next, the bundles of sutures were placed in separate sterile Pyrex glass tubes 2.5 cm. in diameter and sterilized by adding sufficient ethylene oxide to cover the bundle in each tube (fig. 1). After 30 minutes immersion, the ethylene oxide was poured off and the tubes containing the sterilized fascia were stored in a deep freeze at −10 C. The frozen tubes of fascia then were subjected to the freeze-drying procedure according to the technic developed by Creech.[2] At the end of this procedure, the fascial strips were almost completely dehydrated and in the vacuum in which they were preserved in the sealed glass tubes would last indefinitely without deterioration. To reconstitute the fascia the dehydrated strips with needles attached were removed from the sealed tube and placed in sterile normal saline solution for 5 minutes. The reconstituted fascia then had the appearance and texture of fresh fascia; none of its physical characteristics having been altered by the lyophilization process. It then was ready for use as a suture.

It was not found necessary to use sterile technic in obtaining fascia in the autopsy room as repeated cultures have shown that soaking the strips in saline solution with penicillin and streptomycin and treating them with ethylene oxide just prior to lyophilization achieves sterilization. Dispensing with the need of a sterile technic in securing the fascia greatly facilitates the process of removing it under the conditions normally present in the autopsy room.

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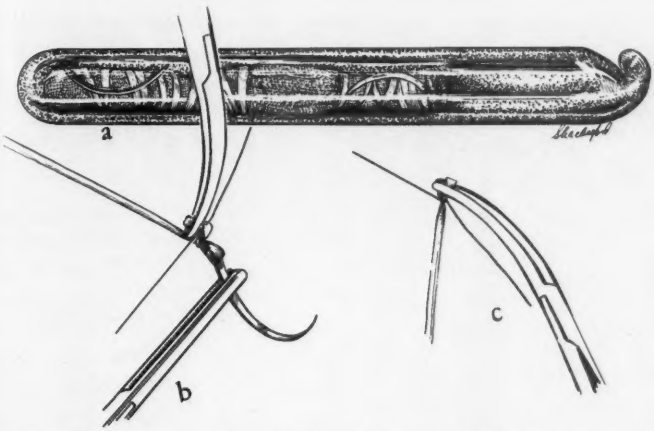

FIG. 1. a, Tube of lyophilized fascial strips with needles attached. b, Securing fascial strip to Gallie needle with no. 36 steel wire. c, Tie at free end of strip. Steps b and c accomplished before lyophilization. (Surgery, July, 1954)

TECHNIC OF HERNIA REPAIR

A somewhat different technic has been used for repairing incisional hernias than that described by Gallie, and we believe that it affords a much stronger approximation of tissues than does the usual single strand continuous suture. Two sutures are started at one end of the wound simultaneously, fixing the end of each suture with a slip knot as shown in the illustration (fig. 2). The surgeon uses one suture and his assistant the other. Each employs a continuous over and over suture, alternating stitches with his partner and producing a crisscross effect somewhat similar to the lacing of a shoe. The ends of the two sutures are tied together with a triple square knot and the knot transfixed with a tie of no. 36 steel wire. Another pair of sutures then is placed and run in a similar manner and so on until the defect is closed. With this technic the edges of the hernia can be approximated in the majority of incisional hernias even though under considerable tension. The large, flat fascial strips will not cut through the tissues and the *shoe-lace* technic affords a much stronger closure than can be accomplished with single strands. Additional reinforcing rows of fascial strands then can be added. It is important that the solidarity of the hernial ring not be disrupted by attempting to dissect out layers of peritoneum or fascia for separate closure. The hernial sac can be left adherent to the subcutaneous fat and obliterated by interrupted sutures of plain catgut. Silk and cotton are best avoided in fascial repairs because of their tendency to cause draining sinuses. A subcutaneous drain for 24 to 48 hours, and a pressure dressing of mechanics waste and Elastoplast lessens the likelihood of a seroma or hematoma forming in unobliterated dead space, particularly in obese patients.

CLINICAL STUDIES

Since July 1953, 63 hernias have been repaired with the lyophilized human fascia using the technic described. All of these patients have been followed-up

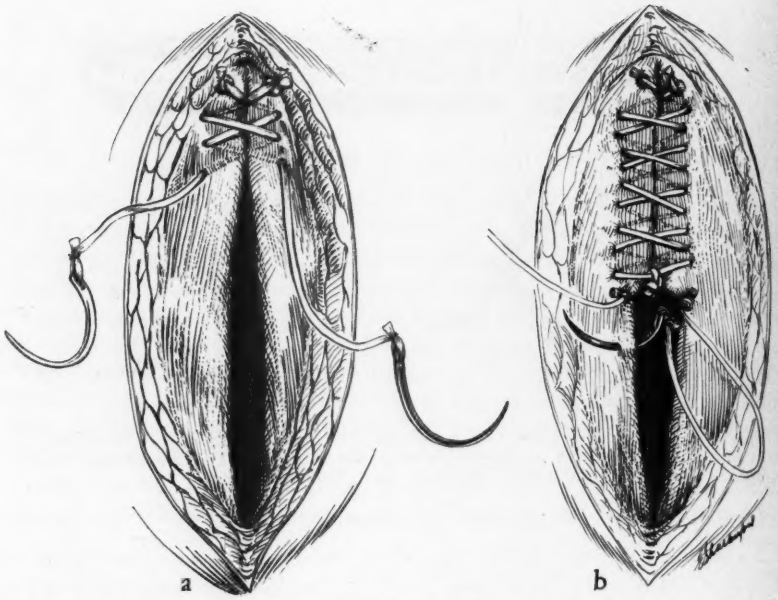

FIG. 2. Technic of repair of an incisional hernia. Edges of the hernial ring are approximated without dissection of peritoneum or fascia. a, Free ends of each strip anchored with a slip knot and a *shoelace* stitch used, the surgeon and his assistant alternating stitches. b, Ends of the sutures tied together with a triple square knot and transfixed with a tie of no. 36 steel wire. A second pair of fascial sutures started. (Surgery, July, 1954)

TABLE I

Hernia repair with lyophilized human fascia lata

Results of repair with lyophilized fascia lata in 63 patients

July 1953 to September 1954

Incision	Cases	Recurrences	Wound Infection
Upper midline	25	1	1
Lower midline	14		
Midline (xiphoid to pubis)	1	1	
Upper right rectus	5		
Lower right rectus	10	1	
Transverse	6	1	1
Subcostal	2		
	63	4	2

and in the 63 patients listed above there have been four recurrences and two infected wounds (table I). All four recurrences have been repaired and the patients have had no further trouble. There was one death in the series, due to a pulmonary embolus on the seventh postoperative day.

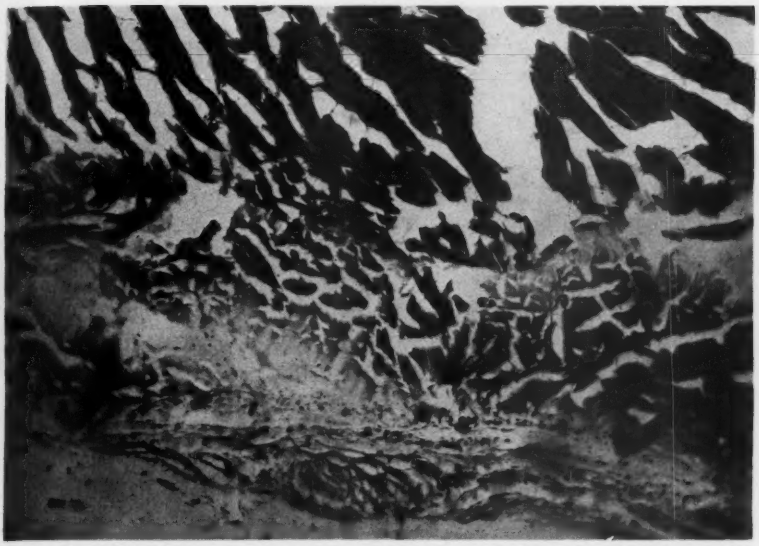

FIG. 3. Lyophilized human fascia lata used to close wound of patient with carcinoma. Had been in place six weeks before death. Fascial suture in upper portion of photograph, host tissues at bottom. No inflammatory reaction noted.

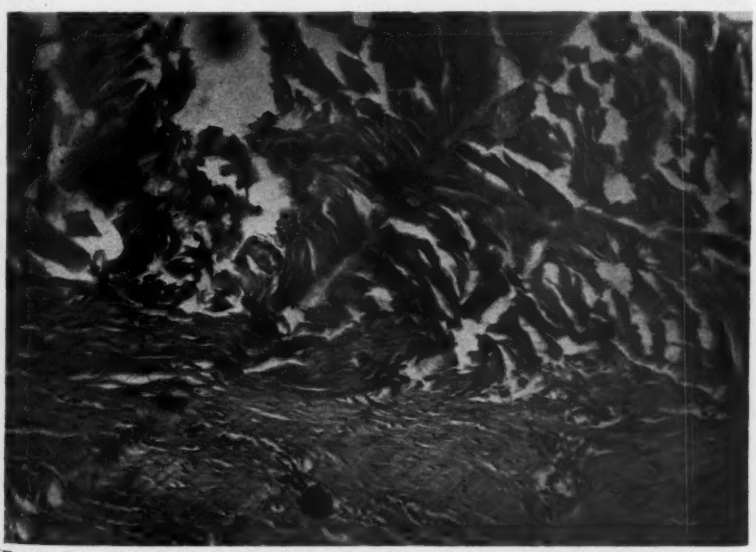

FIG. 4. Lyophilized human fascia lata in place two months before death. No foreign body reaction. Note firm bondage of suture to host tissue.

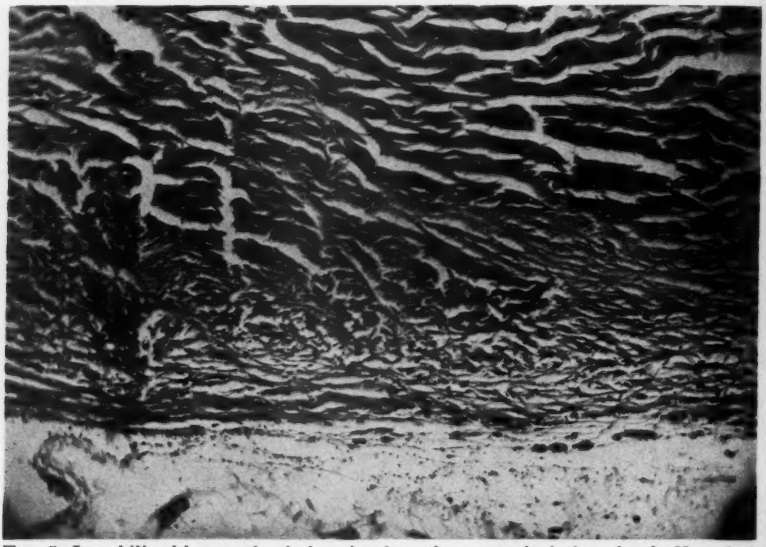

FIG. 5. Lyophilized human fascia lata in place three months before death. Vascularization of the outer border of the fascia is present and there is no foreign body reaction.

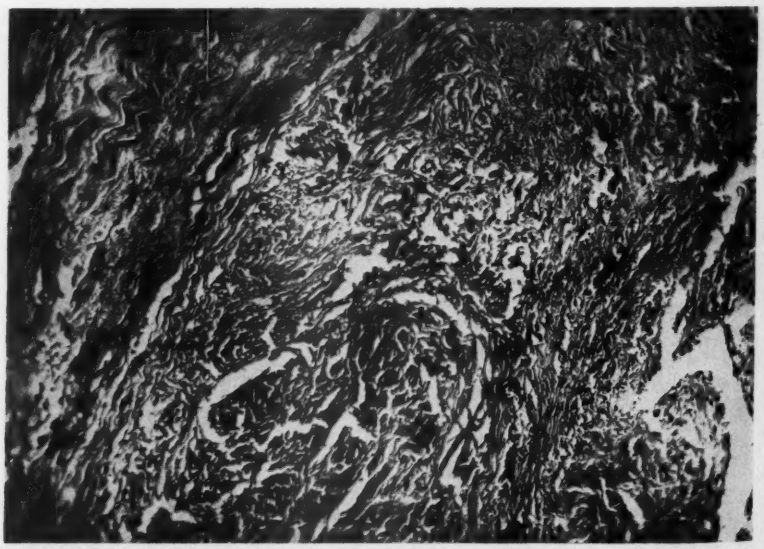

FIG. 6. Lyophilized human fascia lata in place five months before death. Fascial suture in upper left and a second suture in lower middle portion of photograph. Note blending of suture with host tissue.

HISTOLOGIC STUDIES

In order to study the reaction of the host tissues to the implanted homologous fascia a group of 25 patients with carcinoma of the stomach, colon, or pancreas was selected. All of these patients had exploratory laparotomies and in each instance freeze-dried fascia lata was used to close the wound, using the technic described. Some of the patients had palliative resections, and others were non-resectable. All were judged to be *incurable.*

Clinically the wounds healed satisfactorily in all of these patients and they were followed as out-patients. When eventually they died due to their malignancy, efforts were made to obtain autopsies. Permission was granted in 14 of the 19 patients who died, and the wounds of these patients were excised en bloc for histologic study. The period of implantation of the fascia varied from one week to one year in these 14 patients.

All of the sections studied showed firm union of the fascial strip to the host tissue with no evidence of foreign body reaction. In none of the specimens was there any evidence of absorption or dissolution of the fascia (figs. 3, 4, 5, 6).

In 2 of the patients, coming to autopsy, strips of the implanted fascia were dissected out and tested on a tensiometer. In one specimen the fascia had been implanted for a week, in the other for 37 days. In each instance the strip had a tensile strength of 25 pounds which is the average tensile strength of the reconstituted freeze-dried fascia.

SUMMARY

Lyophilized (freeze-dried) human fascia appears to offer the full advantages of fresh autogenous fascia for hernia repair with none of the disadvantages attendant upon securing fascia from the patient's thigh.

Satisfactory clinical results in 63 patients in whom lyophilized human fascia lata has been used with only four recurrences appears to warrant further investigation on this type of suture material.

Histologic studies of the wounds of 14 patients with carcinoma whose incisions had been closed with the lyophilized fascia showed no foreign body reaction to the lyophilized human fascia and tensiometer tests showed no decrease in the tensile strength of the implanted fascia.

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SURGICAL TREATMENT OF EXTENSIVE LYE STRICTURES OF THE ESOPHAGUS IN CHILDREN: CASE REPORTS

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Strictures of the esophagus caused by the ingestion of caustic agents present a difficult problem. Their care has been divided between the otolaryngologists and the surgeons. The otolaryngologists have patiently and persistently used various technics of dilatation in an effort to keep a patent lumen in the scarred esophagus. Their accepted form of treatment has been dilatation within a few days after the burn and then periodic dilatation thereafter until a satisfactory result has been obtained. This method of treatment is known as the "Salzer Method".[5]

There have been many recent developments in the field of esophageal surgery. New studies and technics make it necessary to completely re-evaluate the entire treatment of the caustic burn of the esophagus.

The pathology as described by Burford, Webb, and Ackerman[3] and Bosher, Burford and Ackerman[2] is such that one would not expect a permanent cure in the majority of the patients if they were treated by dilatation alone. Burford, Webb, and Ackerman state that the destruction to the epithelium takes place within the first few hours following the burn. There is edema and congestion of the entire esophageal wall. This is followed by areas of mucosal gangrene and even sloughs of the entire mucosa and submucosa. Bacterial invasion takes place within 24 hours and extends well into the muscularis. The destructive process is most severe in the lower portion of the esophagus. The healing process is slow. Re-epithelization does not become complete before six weeks, and the inflammatory process has not completely subsided until some time after the epithelization is complete.

Early dilatation, therefore, must carry considerable risk, both as to perforation and toward further injuring the already edematous, congested esophageal wall. Furthermore, it is a well-known fact that if a stricture is fully developed, it rarely responds to repeated dilatations.

The earliest studies in esophagogastrostomy were reported by Biondi[1] in 1895. The early literature seems to indicate that everyone had difficulty with leakage at the site of the anastomosis. In addition to that, many had difficulty with stricture formation in the same area. In 1923 Miller and Andrus[6] excised a circular area twice the size of the lumen of the esophagus from the fundus of the stomach, and in their series they reported no strictures. Carter, Stevenson, and Abbott[4] studied esophagogastrostomy in 52 dogs and reported their findings in 1941. They were interested particularly in perforation at the suture line and in

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stricture formation. In their series no strictures followed the end to side type of anastomosis when one end of the esophagus was anastomosed either to the edges of the incision in the fundus of the stomach or to the edge of the circular area excised from the fundus. Their incidence of perforation at the line of anastomosis was 89 per cent when no anchoring sutures were placed in the stomach or esophagus. Their incidence of perforation at the line of the anastomosis was none when both the esophagus and the stomach were anchored in position. This anchoring of the esophagus and the stomach appears to be an important technical step.

CASE REPORTS

The surgical procedure we employed was similar to that employed by other writers. Our series is small. We found 2 six year old girls in the program of the Crippled Children's Division who had ingested lye at the age of 2 years. These girls, early in the course of their illness, had vigorous attempts at dilatation. Both had early gastrostomies to aid in the dilatation, and then later these gastrostomies were utilized for feeding purposes. As one would expect, growth and development were below normal. In one child the esophagus had been transplanted to the skin of the neck in the hope that a skin tube could be formed to serve as an esophagus (fig. 1). This tube had never functioned properly, consequently, the attempt was abandoned.

SURGICAL PROCEDURE

A one stage operation was planned, and we approached the stomach and esophagus through a combined incision through the seventh left interspace. The gastrostomy opening was closed first. The diaphragm was split, and the

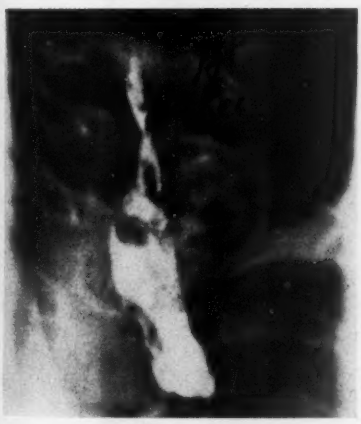

FIG. 1

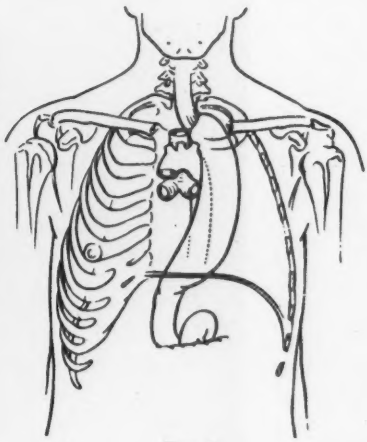

FIG. 2

FIG. 1. Preoperative roentgenograms of case I with barium in the esophagus. The distal outlet of this esophagus was in the skin above the left clavicle.

FIG. 2. Drawing to give general idea as to position of stomach and esophagus following surgery.

stomach was freed to the pylorus. The stomach was mobilized by section of the gastrolienal, gastrocolic, and gastrohepatic ligaments, and the vessels contained in them. The left gastric artery was divided close to its origin, and the left gastro-epiploic vessels were removed. The right gastric and right gastro-epiploic arteries were the only ones remaining to supply the blood to the stomach. The pylorus was mobilized as was the first portion of the duodenum by incision of the peritoneal covering of the lateral margin. A pyloroplasty was done. A second incision was made on the neck and high on the chest on the left. The clavicle was sectioned and the first rib was removed subperiosteally. The esophagus was located and divided as low as possible in the neck. In 1 patient there was not more than 1 centimeter of esophagus present. In the other, the lumen of the esophagus was closed at the level of the sixth cervical vertebra. The stomach was brought up posterior to the hilum of the lung. A circular area of the fundus of the stomach was excised, and a two-layered anastomosis was made using interrupted sutures, throughout. Catgut was used on the inner layer in mucosa to mucosa sutures,

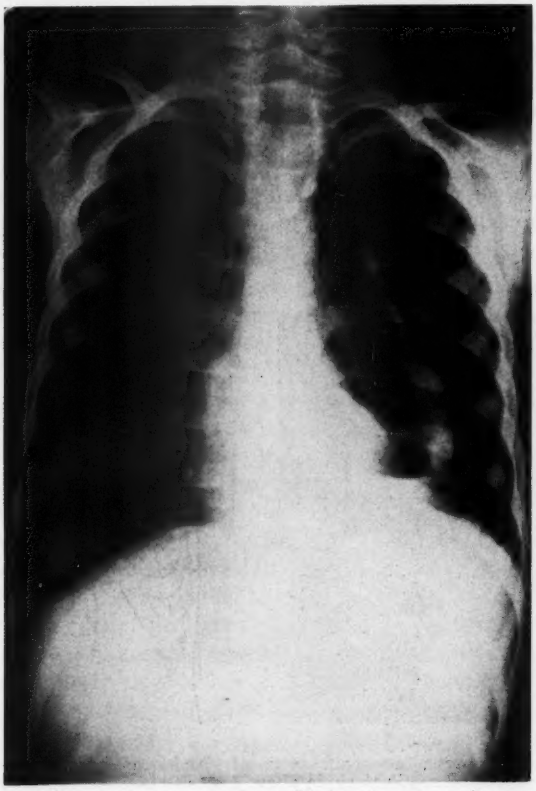

FIG. 3. Postoperative roentgenogram of case I taken two years after operation. No evidence of obstruction. Clavicle appears to have regenerated.

and silk was used on the outer layer which approximated the serosa to the serosa. The stomach was anchored carefully to the posterior cervical tissues and also anchored at several places in the chest to the paravertebral tissues. The esophagus was so densely adherent to the structures about it in the mediastinum that it could not be removed. Both ends of the esophagus were closed first with interrupted sutures of fine catgut, then with a second layer of silk. The diaphragm was closed in a routine fashion, and the level of the diaphragm lay at the pylorus (fig. 2).

FOLLOW-UP

The postoperative course in 1 patient was very smooth She was discharged from the hospital 14 days after the operation and has been checked several times since then. There is no evidence of stricture, and she has made a very normal psst-operative recovery (fig. 3). At the present time roentgenograms of the chest reveal a perfectly clear chest with no widening of the mediastinal shadow.

The other child had a stormy postoperative course, principally because she pulled the decompression tube from her nose a few hours following completion of the operation. We were unable to replace the tube in the stomach. Consequently, she aspirated some vomitus and developed atelectasis. She was kept in the hospital approximately eight weeks following the operation and was dilated just before she was discharged. She has been readmitted on several occasions, and the anastomotic site has been checked. At the present time there is no evidence of stricture formation (figs. 4, 5).

Neither child has had any esophagitis, regurgitation, nor is there any delayed

FIG. 4. Preoperative roentgenogram of case II showing level of obstruction

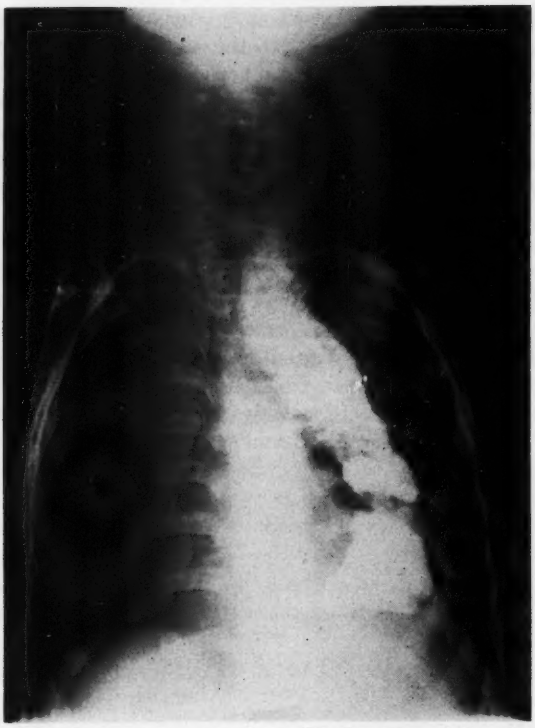

FIG. 5. Postoperative roentgenogram of case II, showing position of stomach and no evidence of obstruction at anastomosis which is in the neck. The stomach was empty in one hour.

emptying time of the stomach. The growth and development have improved considerably since operations.

DISCUSSION

The management of esophageal strictures has changed markedly in the post few years. This has been brought about by a study of the pathologic material taken from the experimental animal and by the well established fact that it now is possible to safely do an esophagogastrostomy in a one stage surgical procedure.

Absolute rest of the damaged esophagus and the use of antibiotics is the preferred treatment early in the course of the disease. The acute inflammatory reaction and the necrosis which may extend well into the muscularis condemns early dilatation and manipulation. Endoscopic examination should be done at the end of four weeks. If a structure is found, cautious dilatation may be tried, for some minimal burns may respond to a few dilatations. However, if the lesion does not respond readily, resection and restoration of the normal swallowing mechanism should be resorted to at an early date.

Patients who have old established strictures should be treated surgically with no undue delay. If a gastrostomy is present, examination with a cystoscope in the stomach is important. Esophograms are an aid in determining the level of resection. In one of the patients it is possible to see the suture line of the anastomosis by laryngoscopic examination.

It is advisable to remove the diseased esophagus, but should this be technically impossible, it can be left in place.

SUMMARY AND CONCLUSIONS

It is possible to restore safely the normal swallowing mechanism in a one stage surgical procedure, even if the fundus of the stomach must be brought into the neck.

Children so treated do not develop anemia nor have any significant retardation of growth and development.

It is, of course, desirable to remove the diseased esophagus, but in cases in which this is unduly hazardous to the life of the patient, leaving the esophagus in will not necessarily result in additional surgery.

Strictures will not develop at the site of the anastomoses, nor should the anastomosis leak in a well performed surgical procedure.

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PLASMA CELL MASTITIS: A REVIEW AND REPORT OF A CASE

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Plasma cell mastitis is a rare condition of the female breast which simulates mammary cancer. It has been described only within the recent past. Adair[2] called attention to this condition with his publication of 10 cases in 1933. He described it as a clinical and pathologic entity. Not all subsequent recorders of this disease have been inclined to accept this premise.[16, 22, 23]

The rarity of plasma cell mastitis is attested to by the paucity of case reports. The largest series is that reported by Cromar and Dockerty,[9] who found 24 cases in 12,000 breast cases at the Mayo Clinic. Parsons and associates[21] found 5 cases in 1500 specimens from female breasts over a period of six and one-half years. Cassie[6] reviewed the English literature and was unable to find any reports indigenous to the British Isles prior to the case reported by him in 1953.

It has been suggested that the relative obscurity of plasma cell mastitis is dependent upon a lack of recognition of the disease and a failure to record those cases which are diagnosed.[19]

HISTORY OF THE DISEASE

Cutler[12] has recorded some interesting aspects of the history of this disease He relates that Ewing in 1925 suggested the name *"Plasma Cell Mastitis"* for a series of cases of inflammatory disease of the breast, characterized by infiltration with plasma cells. Cutler, who was associated with Ewing at Memorial Hospital in New York, reviewed 10 cases at that institution and further studied them. These cases were reported by Cheatte and Cutler in 1931, in their book on "Tumors of the Breast".[7] This constitutes the first report of the disease by the title *Plasma Cell Mastitis.* The following quotation from Payne and associates[22] is interesting: "Although the latter term (plasma cell mastitis) was first used by Ewing, he has never claimed priority of authorship which he attributes to Adair".

Cutler suggested that the clinical features of plasma cell mastitis were reported by Courtin in 1899 in a case report before the Bordeaux Medical and Surgical Society. Coquet, in discussing the case, related his observations of a similar case of an inflammatory lesion of the breast with the signs of malignancy subsiding after local applications. No histologic verification of these cases exists. Cromar related that the disease was reported first from Europe by Ingier as mastitis obliterans in 1909; and that Hoerz reported another case in the ensuing year by the same title. Cromar further stated that the disease was reported as a *"localized lymphogranuloma"* by Kuchens in 1928. Cassie and Cutler asserted that the case report by Hoerz may have been a case of fat ne-

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TABLE I

Number of cases of plasma cell mastitis reported

Author	Year	Number of Cases
Adair	1933	10
Cutler, M.	1933	2
Moore	1933	1
Rodman et al.	1939	1
Miller	1939	1
Frank	1939	3
Cromar et al.	1941	24
Parsons et al.	1941	5
Payne et al.	1943	2
Gaston et al.	1947	3
Halpert et al.	1948	1
Cutler, M.	1949	1
Cromar	1949	3
Manoil	1952	1
Cassie	1954	1
Ferrara	1954	1
		62
Gronwald (9)	1931	1
Schultz (6)	1933	10
Lubschitz (12)	1943	1
Mortara (18)	1950	2
		14
		76

crosis. Thereafter, the disease has been reported as plasma cell mastitis and/or mastitis obliterans. Adair's detailed report of 10 cases in 1933 has stimulated continued interest in the disease. These are the same cases discussed in the book "Tumors of the Breast" by Cheatle and Cutler. In the American and British literature, there have been 62 cases reported formally, or mentioned in discussions. See table I for these cases. Also listed are 14 cases from other sources whose authenticity is accepted by the indicated reference.

NATURAL HISTORY OF THE DISEASE

There is general agreement that plasma cell mastitis is an inflammatory lesion of the female breast. Two phases of the disease have been described by Adair.

1. The Acute Phase: This is characterized by signs of inflammation in the breast which subsides after several weeks to a month. The symptoms may vary in intensity; inflammation may be so mild as to be forgotten by the patient; and the history of inflammation is elicited with difficulty. There may be chills and fever. The breast may be tender, swollen and reddened. A creamy nipple discharge is frequent. The extent of the inflammatory process in the breast is variable but usually more than one quadrant is involved. The axillary nodes

usually are enlarged. As the inflammatory reaction subsides, the breast becomes less red in color; less swollen, somewhat nodular and with better localization of the process. This latter occurrence has been referred to as the subacute phase.[6, 13]

2. The Residual Phase: All signs of inflammation have subsided. A discrete, firm, well-defined, deep-seated mass is present in the breast, which varies in size. Skin fixation may be present. Fixation to the chest wall may occur. The mass may be painful. Nipple discharge, which usually is creamy and thick (and may be bloody), is common. Nipple retraction is frequent. The axillary nodes frequently are palpable. The clinical picture is that of carcinoma. The residual mass may be present for several years before excision, or it may completely disappear without treatment.

It is a disease of parous nonlactating women. The majority of cases occur in the third, fourth and fifth decades. It has occurred in elderly women. Adair stressed the importance of a history of inflammation in making the diagnosis. On the basis of history and clinical findings, he diagnosed 2 cases which were followed for two years before excision was done. Histologic study confirmed his clinical impression in both cases.

The etiology of this interesting condition is not definitely known. There is general agreement that the lesion is inflammatory. Most observers believe that the cause is chemical rather than bacterial. This opinion was expressed by Ewing[14] in his original description of the disease.

Rodman and Ingleby[23] suggested a causal relationship with the products of lactation and have produced similar lesions by injection of pancreatized milk into the breasts of virgin rabbits, and by the injection of pancreatic extracts into the breasts of lactating rabbits. The cause seems to be a periductal extravasation of some fatty material, probably fatty acid crystals in milk, from the ducts into the periductal fibrous tissue. Nipple retraction probably is a contributory factor, presumably because it prevents efficient drainage of milk from the breast. In approximately half of the cases reported in the literature, nipple retraction was found.

Bacterial studies have been disappointing. Cultures have revealed no significant organism. Guinea pig inoculations of excised material have been fruitless. If it is of infectious etiology, Cole[8] has suggested a bacterum of low virulence or a virus. There is nothing to substantiate this. He also suggested a causal relationship to fat necrosis, which other authors have also proposed.[1, 17, 22, 23]

Parsons and associates[21] have suggested that focal infection, such as tonsillitis, may cause ulceration of duct epithelium in the breast with extravasation of duct contents into the periductal tissue and the production of the plasma cell mastitis picture. One of these authors observed a patient with mastitis who improved after a tonsillectomy had been done.

PATHOLOGIC ASPECTS

Adair described these outstanding pathologic characteristics:

1. The presence of the plasma cells in the breast occurring in broad sheets and infiltrating ducts, duct walls and interstitial tissues.

2. The proliferation of duct epithelium piling up 6 to 10 rows deep.
3. The formation of giant cells by the heaped-up lining cells.

The histologic appearance may resemble comedocarcinoma due to the proliferation of duct epithelium. It also resembles tuberculosis, but there is no caseation in plasma cell mastitis.

Ewing has described the process as follows: "From a survey of the present material, it appears that we are applying the term 'plasma cell mastitis' to a special group of chronic or subacute suppurative inflammation of the duct system of the breast. These cases differ from the ordinary suppurative mastitis in the absence of definite abscesses and generally in the wide extent of the process. The process also is less acute and the productive element much more pronounced than in suppurative mastitis. These facts indicate that bacterial infection plays a less prominent role and chemical irritation a more prominent role than in suppurative mastitis. In the particular group of cases which has attracted our attention, the plasma cell infiltration is extremely abundant and widespread, producing rather bulky tumor masses which clinically resemble active carcinoma and even under the microscope may be difficult to distinguish from cellular carcinoma.

The main gross anatomic feature is the presence of many greatly thickened ducts which are filled with puriform material, and may extend over a large segment or nearly the whole of the breast. In many characteristic cases, the cellular exudate is diffuse, making a broad, opaque, sometimes yellowish tumor-like mass in which distended ducts are less obvious or not even visible. These are the cases which resemble carcinoma, but as a rule, there are small foci of puriform softening which are not found in carcinoma."

"The plasma cell exudate begins in the walls of the ducts and extends between the acini in adjoining lobules where the process becomes diffuse. Polymorphonuclear leukocytes are present in variable numbers, but often are very scanty. The phagocytosis of fat is a prominent feature and many of the cases show a great many large plasma cells engorged with fat and resembling xanthoma. These areas have a yellow color."

"Proliferation of the lining epithelium is a peculiar and prominent feature. The affected ducts are lined by from 6 to 10 rows of large, somewhat hyperchromatic, epithelial cells which often raise the suspicion of carcinoma of the duct. Yet, the later progress of these proliferating cells ends not in carcinoma, but in generation, fragmentation and formation of giant cells of all sizes. Many of the giant cells and degenerating epithelium produce structures which closely resemble tuberculosis. Usually the foreign nature of these giant cells is obvious because usually they contain much fat. Stains for tubercle bacilli are negative, and guinea pig inoculations also are negative. The appearance of the proliferative epithelium in the early or milder stages of some cases strongly recalls the appearance of comedocarcinoma. In a few cases, the proliferating epithelial cells produce many small hyperchromatic giant cells, a picture which again approaches that of large cell carcinoma."

There are no histologic characteristics of involved axillary lymph nodes. Non-

specific hyperplastic inflammatory changes usually are present. Plasma cells may or may not be found.

TREATMENT

The treatment of plasma cell mastitis is similar to that of other breast tumors. A biopsy section is studied by frozen section. If the mass is diffuse, as it may be in the inflammatory stage, a biopsy study without excision would seem to be adequate. If the mass is well defined, excisional biopsy should be done. If there is a reasonable doubt as to whether the specimen is plasma cell mastitis or a plasmocytoma, or a carcinoma, the wound should be closed and paraffin sections should be made. Depending upon the results of these paraffin sections, simple excision of the mass would suffice, or radical mastectomy will be necessary. If the breast is the seat of multiple masses, simple mastectomy is preferable. If the axillary nodes are large, these can be excised easily through the simple mastectomy incision.

DISCUSSION

Some aspects of plasma cell mastitis which have been propagated by past recorders of this disease confuse the reader. It is described as a disease of nonlactating parous women. Yet, in 10 cases described by Adair, there was 1 of the 10 who was lactating at the time the diagnosis was made. Our only contact with the disease thus far has been in a young woman who was three weeks postpartum.

It has been stated by some authors that previous pregnancy is a requisite to making the diagnosis. Cromar, in 1949, in his correspondence in the British Medical Journal, states, "It has been observed only in parous women . . . lactation has preceded every reported lesion." However, in 1933, Cutler wrote, "I have recently observed a case of plasma cell mastitis in a woman, age 40, in whom there had been no previous pregnancies." He did not elucidate on this case and reported another case in detail. Similarly, the case report of Rodman and Ingleby is concerned with an unmarried female and there is no mention of pregnancies. One is led to believe that she had never been pregnant.

Two cases of recurrence of the disease have been reported by Parsons and co-workers; these being the only recurrences thus far recorded. One of these cases was described as an abscess in the wound, occurring five months after excision of the original mass. Healing occurred with incision and drainage of "a small amount of thick, yellow pus". A biopsy section of the abscess was not taken. Later authors have concluded that these recurrences are evidence of the malignant potentiality of plasma cell mastitis. This conclusion was not intended by Parsons and associates, who describe the disease as inflammatory and benign. There is reason to expect that other cases of recurrence will be reported since recurrence is not unknown in other benign inflammatory lesions of the breast.

C. W. Cutler, Jr.[11] presented a case report before the New York Surgical Society on Jan. 24, 1934. This case was that of a 49 year old woman, who had an

excision of a breast mass in June 1932. The histologic diagnosis was plasma cell tumor. Thereafter, histologically similar masses occurred in the right vocal cord, left sternoclavicular fascia, chest wall and antrum. This case was discussed by Adair,[3] who said that he and Ewing had examined the slides and concurred in the diagnosis of a malignant plasma cell cytoma or plasma cell sarcoma. As such, it is not related to plasma cell mastitis. It is this case which is mentioned frequently as displaying the malignant potentialities of plasma cell mastitis. Miller[19] summed up this situation as follows: "If the original tumor was correctly diagnosed as plasma cell mastitis, it is an entity capable of metastases. If this is not the case, there is a malignant neoplasm so closely resembling plasma cell mastitis in histopathology as to be markedly confusing". Halpert and associates[17] reported 3 cases of carcinoma of the breast in which there was a predominant plasma cell reaction around the ducts and acini. The plasma cell reaction was distinct from the neoplastic involvement and thought to be independent of it.

Further, there is doubt whether or not plasma cell mastitis is precancerous. Ewing prompted the study of this disease because it was an inflammatory condition, clinically resembling carcinoma, which was being treated by radical operations. He realized that the victims of this disease were being treated with justification, but by needlessly radical operations. In many of the early reports, a significant number of patients were treated by radical mastectomy. The explanation, of course, is obvious. Frozen section had not yet become as generally available, nor as well established, as it is now. Because it mimicked carcinoma, plasma cell mastitis was being treated as a malignancy. Gaston[16] suggested the malignant potentialities of plasma cell mastitis in two ways:

1. As a malignant epithelial neoplasm, as a result of dedifferentiation of the hyperplastic duct epithelium.
2. As a malignant plasmacytoma.

The following quotation is from Cromar and associates, "Plasma cell mastitis has been accused of possessing potential malignancy, but this suspicion has never been justified by experience. Furthermore, there is no clear evidence to suggest that plasma cell mastitis is related in any way to the malignant plasmacytomas, which occur in other parts of the body."

This brings us to another point of controversy. There are those who doubt that it is a separate pathologic entity and assert that it is poorly named. The similarity with various other benign conditions was stated by Adair. Of 11 diseases mentioned by him, 7 were various forms of mastitis. Rodman and Ingleby remarked about the similarity of such conditions as plasma cell mastitis, infected galactocele, lactation mastitis, and fat necrosis and believed that the same etiologic agent was responsible for all. At least two subsequent authors concur. Payne and associates preferred the term mastitis obliterans to plasma cell mastitis. They declared that plasma cells are present, histologically, in a variety of conditions in which chronic inflammation is a feature. Thus, the plasma cell is distinguishing of no particular disease. In the 2 cases reported by them, plasma cell infiltration was not a characteristic feature. Yet, these cases are accepted as cases of plasma cell mastitis by Cutler, who helped establish

plasma cell mastitis as a distinct entity. The histologic difference between plasma cell mastitis and mastitis obliterans is one of degree. Plasma cell mastitis is characterized by moderate to marked infiltration with plasma cells. In mastitis obliterans, there is a pronounced occlusion of the ducts by granulation tissue and plasma cell infiltration is less prominent. The clinical course of both diseases is similar, as it is in other inflammatory lesions of the breast. While plasma cell mastitis may be a separate pathologic entity, conversely, it may be but a variation of other forms of mastitis. Current textbooks of pathology continue to treat it as a separate entity.[1, 4, 5] However, Ackerman[1] stated, "We have been impressed by the similarity between plasma cell mastitis, fat necrosis, and mammary duct ectasia. We believe that they are all essentially the same process and only vary in degree."

CASE HISTORY

Mrs. S. M. was admitted to the Infirmary, U. S. Naval Air Test Center, Patuxent River, Maryland on Nov. 15, 1953. This 27 year old, white woman, gravida IV para IV, was seen on Nov. 12, 1953 with the complaint of a swollen right breast. She was delivered of a normal child on Oct. 24, 1953. She did not nurse the baby, and at no time was there enlargement or engorgement of the breast. Several days following the birth of her child, soreness and redness of the skin of the breast was noted. She was seen by another physician who treated her with local applications and daily penicillin injections for several days. On this regime the inflammation partially subsided, but there remained a firm movable mass, 3.5 cm. in diameter, in the lower medial quadrant of the right breast, with minimal local heat and redness of the skin overlying the mass. On examination, this mass was not painful, and

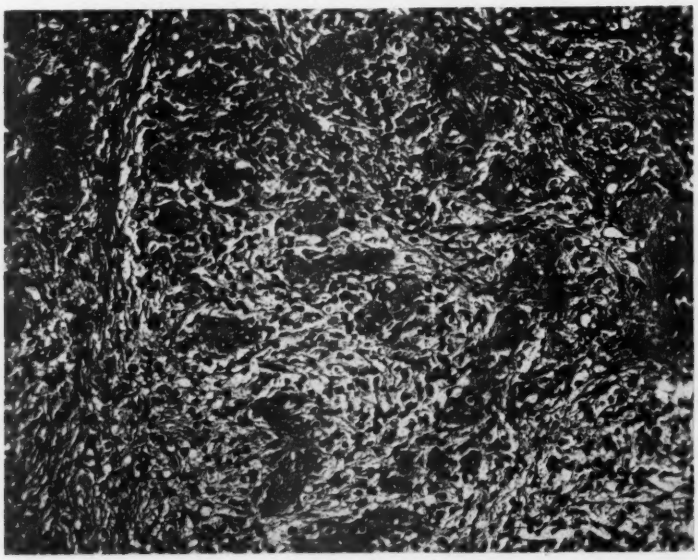

FIG. 1. High power magnification showing interstitial inflammatory reaction in a breast lobule. Cellular component is characterized by multiplicity of plasma cells.

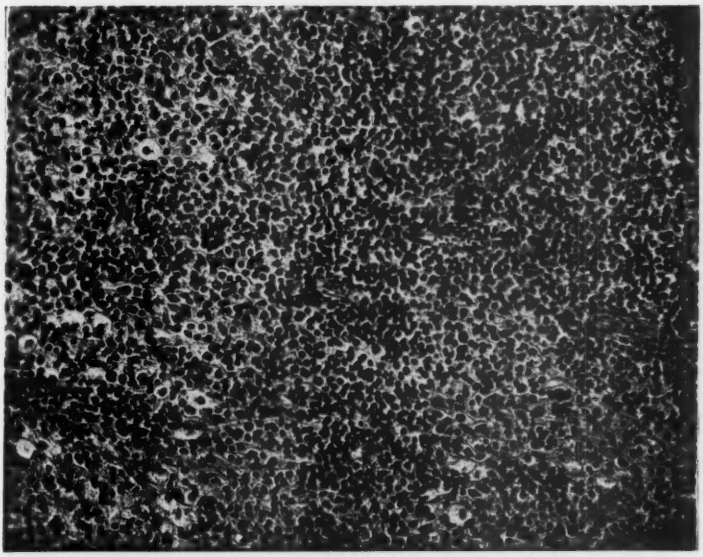

FIG. 2. High power magnification demonstrating the intense cellular infiltration with polymorphonuclear leukocytes, lymphocytes and plasma cells.

milk could be expressed from both nipples. There seemed to be some fixation to the chest wall. In addition, there was a thick, yellow discharge from the right nipple. There was no nipple retraction. There were no masses in the opposite breast. No axillary nor supraclavicular nodes were palpable. On Nov. 16, 1953, the mass was excised. The diagnosis of plasma cell mastitis was made after study of the specimen by the Pathology Department, U. S. Naval Hospital, Bethesda, Maryland (figs. 1 and 2).

CONCLUSIONS

Plasma cell mastitis is a benign inflammatory disease of the breast. It has been described as a distinct clinical and histologic entity. However, its resemblance, both clinically and histologically, to a variety of other inflammatory diseases of the breast is more than coincidental. Apparently, there is no relationship to malignant plasmacytoma. The treatment is simple excision, after malignancy has been excluded.

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CONGENITAL DIVERTICULUM OF THE URETHRA

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The sac of a congenital urethral diverticulum usually arises from the pendulous urethra, and is composed of epithelium and smooth muscle. A congenital diverticulum is differentiated from the acquired type by its appearance at an earlier age and in the absence of infection, trauma, calculus or stricture. The acquired sac may be formed from epithelium alone, and most commonly occurs in the bulbous urethra. Peripheral obstruction is the most important predisposing factor in both congenital and acquired types of diverticula, although apparently congenital diverticula may arise in the absence of obstruction.

Each type of diverticulum is an uncommon lesion in men. Approximately 236 cases of diverticula of the male urethra have been cited in previous reviews.[1, 2, 3, 4] Among these are 96 cases classified as diverticula of congenital origin. In women, acquired diverticula are common, but the congenital type has not been recorded.

The following case report is the ninety-seventh example of a congenital urethral diverticulum found in the literature. The patient is one of the youngest to have survived operation for the condition. A postoperative complication of diverticulectomy during infancy is described.

CASE REPORT

A baby boy, aged 32 days, was examined on July 14, 1953, for progressive enlargement of the penis. This abnormality had been noticed by his mother seven days after delivery. She stated that his penis rapidly enlarged during each urination; decreased in size within a few minutes after voiding, but remained larger than normal. The baby had a small stream but no abnormal incontinence. No other urinary abnormalities were found.

Physical examination was normal except for the penis. On the ventral surface of the penile shaft was a fluctuant, slightly tender mass, measuring 1 by 2 cm., extending to the coronal sulcus. The urethral meatus was pinpoint in size, and urine could be expressed by pressure on the mass.

A urethrogram was obtained by injecting 3 cc. of dye into the meatus with a blunt needle (fig. 1). An excretory urogram showed a normal upper urinary tract. A postvoiding film confirmed the presence of the urethral diverticulum. The blood and urine were normal.

To prevent urinary infection and damage to the upper urinary tract, a meatotomy and diverticulectomy were done on July 17, 1953. A longitudinal incision was made through the median raphe. The diverticulum was dissected free and removed. The defect in the ventral surface of the urethra was 1 cm. long. With a 12F woven bougie in place, the remaining walls of the sac were sutured over the urethral defect. A perineal urethrostomy was made and an 8F catheter was sutured to the skin. The penis was attached to the abdomen with one silk suture through the prepuce.

On the day following operation, a mild diarrhea began. This caused an infection in the urethrostomy wound. On the sixth postoperative day the catheter inadvertently came out, and the patient voided through the distal urethral defect. Disruption of the wound resulted

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FIG. 1. Urethrogram showing congenital diverticulum of the urethra in an infant 32 days old.

in penile hypospadias, which allowed easy voiding by the time of hospital discharge. The hypospadias will be repaired at a later date.

Microscopic examination showed the diverticulum to be composed of stratified squamous epithelium and smooth muscle tissue.

DISCUSSION

The distinction between congenital and acquired diverticula is arbitrary and of little therapeutic importance. Some diverticula appear to be caused by combined congenital and acquired factors. For instance, a congenital external meatal stricture may cause simultaneous diverticulum formation, and acquired peripheral obstruction may cause a diverticulum to form at a congenitally weak point in the urethra. In the absence of a clear-cut history, differentiation of the congenital from the acquired type is facilitated by the location of the diverticulum and by the histologic elements composing its sac.

The pendulous urethra is formed on the ventral surface of the phallus by the union of the urethral folds over the urethral groove. The median raphe denotes the line of fusion. The fusion may be partial, resulting in hypospadias. In some instances complete but so-called weakened fusion occurs. Distal obstruction may increase intraurethral pressure sufficiently to cause diverticulum formation at the point of weak fusion.

The most common cause of urethral obstruction in the fetus is congenital stenosis of the external meatus. This lesion is present in about one-half of the

patients with hypospadias, and has been reported in other cases of congenital diverticula of the urethra. Assuming function of the fetal urinary tract, a congenital diverticulum was formed in this patient as a result of increased intraurethral pressure. Rapid enlargement of the sac occurred as the urinary output increased during infancy.

SUMMARY

A case of congenital urethral diverticulum associated with congenital stenosis of the external urethral meatus is presented. Prompt relief of obstruction by meatotomy and diverticulectomy prevented damage to the upper urinary tract. This case illustrates that diverticulum formation in the fetal urethra may occur as a result of increased intraurethral pressure. During infancy, one complication of urethral diverticulectomy is postoperative hypospadias, which, although necessitating later repair, is less harmful than the diverticulum.

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CALCIFIED PSEUDOCYST OF THE SPLEEN: CASE REPORT

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Since the first splenic cyst was reported by Andral[1] in 1829, a total of 188 cases of nonparasitic cysts of the spleen have been reported. This paper is presented to report the finding of a calcified cyst of the spleen, the thirty-second such case described in the medical literature. Our case does not differ greatly from those already reported except for one interesting detail. This large cyst emanated from the lower pole of the spleen with elevation of the left diaphragm and no depression of the left kidney. The symptoms were chiefly abdominal, but in addition the patient experienced pain in the left costovertebral angle and shooting pains in the left chest.

CASE REPORT

A 43 year old white para ii, gravida ii, was first seen in the clinic on Feb. 15, 1954 complaining of fullness after eating which she had noted three months previously, and which had been gradually increasing. She also noted pain in the left costovertebral angle and shooting pains in the left chest which increased in severity after partaking of large meals. She stated that her appetite seemed to increase and that she had a gnawing sensation in the epigastrium. Her weight decreased from 124 to 111 pounds in two and one-half months. Three weeks before her initial clinic visit, she began to have a definite increase in severity of symptoms accompanied by nausea and vomiting. She lost an additional 20 pounds, and became increasingly nervous. She denied any additional gastrointestinal symptoms except an increase in the size of her stools the past few months. She had increased urinary frequency, some urgency and occasional nocturia.

She claimed two episodes of rheumatic fever, the most recent occurring 15 years ago. She also had had pain down the right leg and weakness of that extremity for the past two or three years. She had been involved in an automobile accident nine years before her present illness. At that time she was forcibly thrown against the steering wheel and was hospitalized for several days during which time she was *weak and sweaty* and suffered severe abdominal pain.

Physical examination revealed a thin, anxious woman with abdominal tenderness in the left upper quadrant and epigastrium with a definite voluntary guarding. There were no palpable masses. There was moderate left costovertebral angle tenderness. The lungs were clear to percussion and auscultation. The heart was normal in size and shape. The blood pressure was 110/68 and the pulse was regular. Murmurs were not elicited. There was hypesthesia of the dorsum of the right foot.

An upper gastrointestinal roentgenologic examination on February 17, showed a well circumscribed cystic mass in the upper left quadrant of the abdomen displacing the greater curvature of the stomach toward the midline (fig. 1). The mass moved slightly with respiration, but did not pulsate. Intravenous pyelograms showed normal kidney shadows with no displacement (figs. 2, 3).

On admission to the hospital March 4, 1954, the laboratory studies were within normal limits.

She was very apprehensive and appeared dehydrated. She was given fluid therapy and

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Presented during the Oklahoma City assembly of The Southwestern Surgical Congress, Sept. 20–22, 1954, Oklahoma City, Oklahoma.

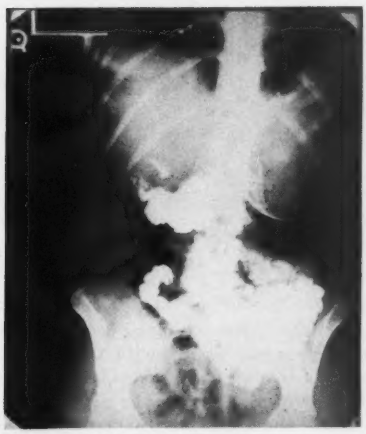

FIG. 1. Photograph of roentgenogram showing greater curvature of stomach pushed to midline by tumor mass.

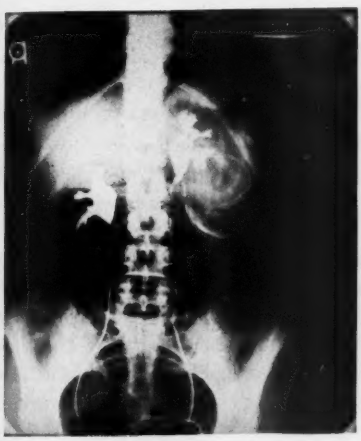

FIG. 2

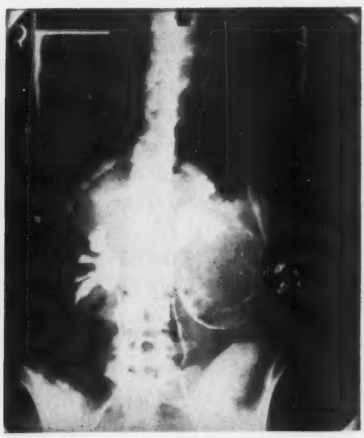

FIG. 3

FIG. 2. Photograph of retrograde pyelogram showing cystic tumor in relation to left kidney. No displacement of kidney downward.

FIG. 3. Intravenous pyelogram showing elevation of left diaphragm and magnified appearance of cyst as compared to figure 2.

protein supplement in preparation for operation. After several days she began taking small feedings and tolerated food somewhat better.

She was operated upon on March 13, through a left paramedian incision. A firm cyst 10 cm. in diameter was found to be in continuity with and replacing the lower pole of the spleen. The spleen and cyst were removed (fig. 4).

Pathologic Report. The gross surgical specimen consisted of a spleen weighing 150 Gm. The lower pole was replaced by a cyst 10 cm. in diameter. The firm wall measured 0.2 cm. in thickness. Microscopically, the sections of spleen appeared normal. Sections through

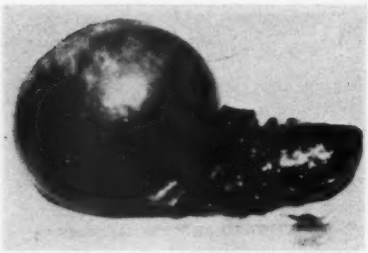

FIG. 4. Photograph of specimen removed at operation. Calcified cyst of lower pole of the spleen 10 cm. in diameter.

the cyst wall showed fragments of splenic pulp and dense fibrous tissue and minute areas of calcification. The inner surface was ragged, with a small quantity of blood attached. Diagnosis: Bulky pseudocyst of the spleen with calcification of the wall.

In addition to the usual postoperative care, the patient was placed on anticoagulant therapy. She progressed rapidly and symptoms disappeared except for occasional episodes of shooting pains in the left chest and left shoulder lasting from 15 to 30 minutes. These became less severe and soon disappeared. Electrocardiograms on two occasions were interpreted as normal.

She was discharged from Masonic Hospital on March 26, 1954 feeling well and fully recovered from her operation. She has been well since (September 1954), and has resumed the full duties of a busy housewife and mother.

DISCUSSION

Cysts of the spleen are extremely rare. They are the least frequently encountered cystic disease involving the abdominal viscera. Pemberton[5] reported 800 cases in which splenectomy was done with only 0.5 per cent showing cyst formation.

Classification has been credited to Fowler[4] who, as early as 1913, summarized the pathologic findings of this disease and advanced the accepted theories. He divided splenic cysts into dermoid, parasitic and nonparasitic cysts. The parasitic form is associated entirely with echinococcic disease, and we will not concern ourselves with this phase of splenic pathology. The nonparasitic class, which is the most frequent, is divided into true and pseudocysts, which are distinguished by the presence or absence of a specific secretory lining.

The incidence of splenic cysts definitely is related to the etiology. Many theories have been advanced as to the cause of nonparasitic splenic cysts, but trauma is the only proved significant factor.[5] For the formation of cysts, trauma must be sufficient to rupture or otherwise damage the parenchyma of the spleen so that a hematoma will form and later degenerate.

The predominance of cysts in young women of the childbearing age is considered to be a prime factor.[6] When splenomegaly is found in any disease, trauma to the abdomen must be considered basic in the formation of a large percentage of splenic cysts.

Cysts of the spleen usually do not cause any symptoms. The ease of diagnosis is in direct proportion to the size of the cyst. Small cysts usually are not dis-

covered except incidentally during an exploratory operation or at the autopsy table. If the cyst is large it usually produces pressure and the predominant symptom is pain. This may only be a feeling of heaviness in the left hypochondrium, but if there is associated peritoneal involvement, there may be severe pain with vomiting and fever.[5] Digestive disturbances often are observed as a result of displacement of the stomach and intestines.

The signs of organ displacement by roentgenogram are displacement of the stomach downward and to the right, elevation of the diaphragm, depression of the kidney and depression of the splenic flexure downward. Duby,[3] quoting Benton, states that the downward displacement of the splenic flexure is practically a pathognomonic sign of a splenic cyst. Splenomegaly may not result in depression of the splenic flexure of the colon.[2] Tumor of the retroperitoneal organs push the colon forward. Pancreatic cysts, kidney tumors and ovarian cysts usually are lower in the abdomen.

The differential diagnosis must be made between cysts of the mesentery, omentum, pancreas, kidney and left lobe of the liver. Calcification of an aneurysm[5] involving the splenic or renal artery may produce roentgenographic evidence like that of a calcified cyst of the spleen. The expansile pulsation and bruit may be the differentiating characteristics.

In the early reports of treatment of cysts of the spleen, marsupialization was the method of choice. As knowledge of the disease and a positive approach to the surgical phase advanced, removal of the spleen has been accepted universally. The results from splenectomy in the treatment of cysts have been good with a very low mortality rate.

SUMMARY

A case of calcified pseudocyst of the spleen in a 43 year old white woman is reported. A total of 188 cases of splenic cyst have been described in the literature, 31 of which have been calcified. This case differs from others recorded in that the cyst arose from the lower pole of the spleen and yet produced an elevation of the left diaphragm and no depression of the left kidney. The symptoms were chiefly abdominal, but there also were chest symptoms. The patient was in the childbearing age and gave a history of direct trauma to the spleen. Treatment consisted of splenectomy with complete recovery.

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STRANGULATED HERNIA, A FIVE YEAR STATISTICAL STUDY*

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The purpose of this paper is to review and analyze all cases of strangulated hernia seen at the Confederate Memorial Medical Center over the five year period from Jan. 1, 1949 through Dec. 31, 1953. The literature of recent years has been reviewed and an attempt has been made to compare results obtained at this hospital with those obtained at other medical centers in the United States. To obtain as accurate a tabulation as possible, all hernia cases at this hospital during the five year study period were reviewed and the operative records were carefully studied to obtain a comparative index as to the frequency of strangulation as well as to establish evidence of strangulation in the studied cases.

All cases of hernias in which a definite statement was made in the record that there was some degree of vascular embarrassment present at time of operation were tabulated as strangulated hernias. Those cases of nonreducible hernias in which the wording of the operative record left any doubt as to the presence of vascular embarrassment of the contained viscera were recorded as incarcerated hernias. One case was a patient who had a strangulated hernia and who was not operated upon, was included, in which there was obvious clinical evidence of an incarcerated hernia with signs of extreme toxicity, generalized peritonitis and free air under the diaphragm demonstrated by roentgenogram. Two other cases of strangulation were included that were demonstrated at postmortem examination.

According to Christopher,[1] the term *strangulated hernia* is applicable to any hernia in which the constriction of the neck of the sac causes interference with the circulation of the contents of the sac. Pressure on the veins is the first result of strangulation and therefore the early changes are venous congestion, edema, and transudation of serum. Later the arterial flow is affected and the bowel becomes purplish, the serosa becomes dull in appearance and a fibrinous exudate forms. Of course when gangrene is complete the bowel is black or gray, lusterless and flabby, and the sac contains foul sanguinopurulent exudate.

The cases of strangulation in this study have been placed in three groups to facilitate better evaluation of results obtained. Group I included those cases in which the bowel or other contained viscera demonstrated venous congestion, edema or transudation of serous fluid. Group II included those cases demonstrating cyanosis, serosal exudate, or sanguinous fluid in the sac. Group III included those cases with frank infarction and with foul-smelling black fluid contained in the hernial sac. Jarboe and Pratt[3] included all cases in their series that showed changes ranging from congestion to necrosis. Laufman and Daniels,[5] however, included only those cases with a definite change in color, corresponding therefore to Groups II and III in this series.

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In a review of cases of intestinal obstruction, Moses[6] described the physiologic changes that occur in strangulation of the bowel. It would be helpful in explaining some of the operative deaths to be presented later to review these changes at this time. As a result of the venous obstruction which precedes arterial occlusion, there is a marked increase of intracapillary pressure which causes a massive transudation through the capillary wall of plasma first, and then of red blood cells. Moses stated that this occurs in terms of liters of fluid into the bowel wall, bowel lumen, and peritoneal cavity and therefore explains the frequent shock encountered in these patients. He believed that the toxin theory of the etiology of this type of shock is without much basis. Laufman,[5] on the other hand, emphasized the bacterial source of toxicity in strangulation obstruction. He stated that if the bacterial factor is controlled adequately, the ill effects of strangulation obstruction are greatly diminished and believed that improvement of results in strangulation obstruction in recent years has been due partially to the role of antibiotics in the control of this bacterial factor. The present series of cases occurred during a period in which antibiotics were in general use and this possibly helps to explain in some degree the favorable results to be presented later.

Moses[6] stated that the literature shows a lower mortality rate from strangulated hernias than from other bowel strangulations. He believed that this may be explained by the limiting envelope of the hernial sac which restricts the quantity of transudate lost by capillary seepage. When the strangulation that occurs in hernial sacs is corrected, the capillary bed which has been injured by both direct trauma and anoxia will be subjected to another sudden increase in intraluminal vascular pressure. Therefore, the seepage of blood and plasma through the permeable vascular walls will continue after operation. He believed that this explains the fact that upon occasion a patient operated upon for strangulated hernia may develop shock for the first time a few hours after surgery, and he recommended that this relapse be anticipated and prevented by the prophylactic postoperative administration of whole blood. This fact should be kept in mind during the presentation later in this paper of at least one of the operative deaths.

There were 663 inguinal hernias seen at the Confederate Memorial Medical Center during this five year study period (table I). It is regretable that all hernias that passed through the hospital probably were not coded so that they could be tabulated, but at least this figure will help to give some idea of the frequency of strangulation and incarceration of hernias in this institution. Of these inguinal hernias, 95 per cent were male, 66 per cent were in the Negro race, and 59 per cent were in the fifth through the eighth decades of life. There were 515 hernioplasties done for inguinal hernias during this period. If the hernia recurred and was repaired a second time during the five year period it was not recounted. Direct hernias represented 12 per cent of the hernioplasties. No record of incarceration or strangulation in a direct inguinal hernia could be found. There were 55 cases of incarcerated inguinal hernias, or 8.2 per cent, and 10 per cent of the hernioplasties done were for incarcerations. There were 43 cases of strangulated inguinal hernias, or 6.5 per cent, and 8.3 per cent of the hernioplasties

TABLE I

Total number of hernias

Type	Total	Percentage						Percentage Each Decade										Total repaired	Incarcerated		Strangulated	
		R	L	M	F	W	C	1	2	3	4	5	6	7	8	9	10		Repaired	Total	Repaired	Total
																			%	%	%	%
Inguinal....	663	65	35	95	5	34	66	16	5	6	7	12	15	20	12	4	0	515	10.7	8.2	8.3	6.5
Umbilical...	153			33	67	13	87	49	2	7	12	13	8	5	2	1	0	121	19	15	9	7.2
Femoral....	30	60	40	37	63	63	37	0	0	3	3	17	27	20	27	0	3	27	18.5	16.7	44.4	40
Epigastric..	39			62	38	33	67	0	8	8	23	23	15	0	20	3	0	29	6.9	5.1	10.3	7.6
Ventral....	20			30	70	45	55	0	0	10	20	20	25	20	5	0	0	15	20	15	13.3	10
Internal....	3			67	33	33	67	0	0	0	0	67	0	33	0	0	0	3	0	0	100	100

done were for strangulation. It is interesting to note that of all the inguinal hernioplasties done in this hospital that 19 per cent were for cases of incarceration or strangulation.

There were 153 umbilical hernias seen during this period (table I). Of these, 67 per cent were in females, 87 per cent were in the Negro race, and 49 per cent were in the first decade of life. Hernioplasties were done in 121 cases. Incarcerated umbilical hernias represented 19 per cent of the hernioplasties and 15 per cent of the total number of umbilical hernias seen. Strangulated umbilical hernias represented 9 per cent of the hernias repaired and 7.2 per cent of the total. A total of 28 per cent of the umbilical hernioplasties done were therefore for incarceration or strangulation.

Only 30 femoral hernias were seen in the time period covered by this study and 63 per cent were in females and 63 per cent were in the white race (table I). The age distribution was significant in that 91 per cent were in the fifth through the eighth decades of life. There were 27 femoral hernioplasties done and 18.5 per cent of these were for incarcerations. Incarcerated femoral hernias accounted for 16.7 per cent of the total number of femoral hernias seen. Strangulated femoral hernias constituted a very significant 44.4 per cent of the femoral hernias repaired at this hospital and 40 per cent of the total seen. More femoral hernioplasties were done for strangulation and incarceration, namely 62.9 per cent, than were done for uncomplicated cases. Koontz[4] reported a much lower incidence of incarceration and strangulation in femoral hernias at the Johns Hopkins Hospital. He reported a 10 per cent incarceration and a 26.6 per cent strangulation rate, as opposed to a 16.7 per cent incarceration and a 40 per cent strangulation rate at this hospital. On the other hand, Peden[7] recorded that at the St. Louis City Hospital more femoral hernia repairs are done for strangulated than for incarcerated or uncomplicated femoral hernias combined. This is a slightly higher incidence of strangulation than the 44.4 per cent noted in this series. Jarboe and Pratt,[3] reporting from the Mayo Clinic, emphasized that when the small bowel is strangulated in a femoral hernia the clinical picture is one of acute intestinal obstruction. They reported that in 61 per cent of their cases of strangulated femoral hernias the small bowel was strangulated and these cases

presented signs of obstruction. At this institution the small bowel was strangulated in 97 per cent of the cases and was associated with some signs of obstruction. There was 1 case in this series with omentum only strangulated with no signs of obstruction. Jarboe and Pratt[3] also reported that a painful groin mass was present in 96 per cent of their cases. There were several cases in this series in which no definite mention of a groin mass was recorded. This finding should serve to remind one to thoroughly inspect the groin areas for possible hernia when confronted with a case of intestinal obstruction. Valuable time probably is often lost, in so far as salvaging strangulated bowel is concerned, when this simple step is overlooked.

There were 39 cases of epigastric hernia in this series; 62 per cent in males, 67 per cent in the Negro race, and 61 per cent in the fourth through the sixth decades of life (table I). A total of 29 epigastric hernia repairs were done with 6.9 per cent of the repairs for incarceration and 10.3 per cent for strangulation. Of the total number of epigastric hernias seen, 5.1 per cent were incarcerated and 7.6 per cent were strangulated.

Ventral hernias, including incisional hernias, were seen in 20 instances; 70 per cent were in females and 55 per cent were in the Negro race (table I). These hernias were rather evenly distributed in the fourth through the seventh decades of life with a total of 85 per cent of the cases in these four decades. A total of 15 ventral hernioplasties were done with 20 per cent for incarceration and 13.3 per cent for strangulation. A total of 33.3 per cent of the cases repaired were for strangulation or incarceration. Incarcerated cases represent 15 per cent of the total number of cases of ventral hernia and strangulated cases represented 10 per cent of the total.

Only 3 internal hernias were found (table I). All of these were diagnosed at operation and presented the clinical picture of intestinal obstruction. All cases exhibited some degree of strangulation. Moses[6] reported 5 internal hernias, all strangulated, in a series of 223 consecutive small bowel obstructions at the Gallinger Municipal Hospital.

During this five year study period there were 74 cases of strangulated hernia in this institution (table II). Of these, 43 cases, or 58 per cent of the total, were inguinal hernias. There were 11 umbilical hernias representing 15 per cent of

TABLE II

Types of strangulated hernia

Type	Confederate Memorial Hospital		Michael Reese Hospital
	Number	Percentage Total	Percentage Total
Inguinal	43	58	62
Femoral	12	16	19
Umbilical	11	15	
Epigastric	3	4	19
Ventral	2	3	26
Internal	3	4	

TABLE III

Duration of symptoms as it affects the condition of contained viscera and duration of symptoms in relation to mortality rate

Hours	Group				Resection		Exteriorization	Operative Mortality	Hospital Mortality	Total Mortality
	I	II	III							
			Bowel	Omentum	Bowel	Omentum				
Less 12	7	12	2	0	2	0	0	0	0	0
12–24	7	5	4	0	4	0	0	0	0	0
24–36	6	1	1	1	1	1	0	0	0	0
36–48	0	1	2	0	2	0	0	0	0	0
48–60	1	4	1	0	1	0	0	1	0	1
60–72	0	1	3	1	1	1	1	1	1	2
72–84	0	0	0	0	0	0	0	0	0	0
84–96	0	0	2	0	2	0	0	0	0	0
96–108	0	0	0	0	0	0	0	0	0	0
108–120	1	1	2	1	2	1	0	2	0	2
Greater 120	0	3	3	2	1	2	0	1	2	3

the total and 12 femoral hernias or 16 per cent of the total. There were 3 epigastric, 2 ventral and 3 internal hernias. These percentage figures roughly agree with those given by Laufman[5] in his report on strangulated hernias at Michael Reese Hospital. He found that strangulated inguinal hernias comprised 62 per cent of the total as compared to 58 per cent in this series, and that strangulated femoral hernias represented 19 per cent of the total as compared to 16 per cent in this series. The remaining types of hernia represented 19 per cent in Laufman's series and 26 per cent in this series.

Laufman concluded that the duration of symptoms in strangulated hernias does not necessarily reflect the duration of strangulation and that the incidence of gangrene, therefore, bears no relation to the duration of symptoms. The findings in this series of cases completely substantiates this conclusion. He found that those patients with viable bowel had symptoms ranging from 2 to 120 hours. An almost identical range of 3 to 120 hours was found in this series. Laufman's patients with gangrenous bowel had symptoms ranging from 5 to 144 hours while in this series, those patients requiring bowel resection had symptoms ranging from 7 to 192 hours. It will be readily noted that there is an almost complete overlapping of time intervals in the cases of viable and nonviable bowel (table III). This unreliability of depending on the time interval to judge viability of hernia contents is even further borne out by those cases of incarcerated hernia in this series in which symptoms ranged from 1 to 5 days without any evidence of strangulation (table IV).

Laufman very definitely demonstrated that even though the time element is unreliable in judging viability of bowel, there is a direct correlation between duration of symptoms and mortality rate. This is very vividly borne out in this study also, but the time element is more favorable in this series in that there is

TABLE IV

Duration of symptoms in incarcerated hernia

Type	Total	Day				
		1	2	3	4	5
Inguinal	55	45	7	1	1	1
Umbilical	23	14	4	1	1	3
Femoral	5	4	0	0	0	1
Epigastric	2	2	0	0	0	0
Ventral	3	3	0	0	0	0
Internal	0	0	0	0	0	0

TABLE V

Duration of symptoms and mortality rate

Time in Hours	Number of Patients Died	Mortality Rate
		%
Less than 48	0	0
Less than 60	1	1.8
Less than 72	3	5
120	2	40
Greater 120	3	37.5

a lower mortality rate per unit of time than was reported by Laufman. He stated that 17 per cent of his deaths occurred in patients with symptoms for less than 24 hours. There were no deaths in this series, in patients with symptoms up to 48 hours. Laufman also stated that the mortality rate in his series, was 5 per cent in patients with a duration of symptoms less than 60 hours and a mortality rate of 58 per cent in patients with a duration of symptoms greater than 60 hours. In this series there were 55 patients with symptoms of less than 60 hours duration with one death and a mortality rate of 1.8 per cent. There were 59 patients with symptoms of less than 72 hours duration with 3 deaths and a mortality rate of 5 per cent. There were 5 patients with symptom of 120 hours duration with 2 deaths and a mortality rate of 40 per cent, and 8 of greater than 120 hours duration with a mortality rate of 37.5 per cent (table V).

All of the deaths in the Michael Reese Hospital series occurred in patients in the sixth through the eight decades of life and these represented 59 per cent of the total number of deaths. In this series all deaths occurred in patients in the fifth through the ninth decades of life and these cases represented 70 per cent of the total. The youngest patient with a strangulated hernia was 2 months of age and there was 1 case of strangulation in a patient 90 years of age (table VI). Laufman also reported that men between the sixth and eighth decades of life represented 83 per cent of the deaths in his series. It is extremely interesting that women between the fifth and eighth decades of life represent 87.5 per cent of the deaths in this series (table VII). This represents then a complete reversal of the sex predominance between the two series and a slightly younger

TABLE VI

Age and mortality rate

Decade	Total	Bowel Resection	Operative Mortality	Hospital Mortality	Total Mortality
1	7	0	0	0	0
2	3	0	0	0	0
3	5	1	0	0	0
4	7	0	0	0	0
5	12	3	2	0	2
6	12	4	1	0	1
7	16	6	0	1	1
8	8	1	2	1	3
9	3	0	0	1	1
10	1	1	0	0	0

TABLE VII

Sex and mortality rate

	Total	Bowel Resection	Operative Mortality	Hospital Mortality	Total Mortality
Male	49	6	0	1	1
Female	25	10	5	2	7

TABLE VIII

Race and mortality rate

	Total	Bowel Resection	Operative Mortality	Hospital Mortality	Total Mortality
White	16	5	2	1	3
Negro	58	11	3	2	5

age group in this series as far as mortality rate is concerned. This sex predominance is even more striking when one considers the fact that women represent only 33.6 per cent of the cases of strangulated hernia. Race seemed to exert no influence on the mortality rate since 78.2 per cent of the cases were of the Negro race and 62.5 per cent of the deaths occurred in Negro patients (table VIII).

When the cases of strangulation are grouped according to the appearance of the bowel, as outlined earlier in this paper, it is evident that no group is free of mortalities, but of course Group III, the group with frank gangrene, has the highest mortality rate. There were 22 patients in Group I with one death and 4.5 per cent mortality rate. There were 28 patients in Group II with 2 deaths and 7.1 per cent mortality rate. There were 24 patients in Group III with 5 deaths and a mortality rate of 20.8 per cent (table IX). As has been stated previously, Laufman's series of cases corresponds to those cases included in Groups II and III of this series. He reported an over-all mortality rate of 12 per cent and this compares favorably with the total of 52 patients in Groups II and III in this series with 7 deaths and a mortality rate of 13.5 per cent. Further comparison of mortality rates will be mentioned later.

TABLE IX

Condition of contained viscera in relation to mortality rate

	Number	Deaths	Mortality
			%
Group I	22	1	4.5
Group II	28	2	7.1
Group III	24	5	20.8

TABLE X

Comparative bowel resection mortality rates

Confederate Memorial Hospital	6.3%
Michael Reese Hospital	15.8%
Johns Hopkins Hospital	37.5%
University of Pennsylvania Hospital	19%

There were 16 patients requiring resection of bowel in this series, one in which the transverse colon was exteriorized, and 5 in which gangrenous omentum was resected (table III). The case in which the transverse colon was exteriorized resulted in an operative death. None of the 5 patients whose omentum was resected died and in 1 of these, the appendix also was removed because of acute suppurative appendicitis occurring concomitantly. Of the 16 cases of bowel resection, 14 were small bowel, 1 was cecum, and 1 was a Meckel's diverticulum. There was one operative death in those patients in whom the bowel was resected, resulting in a resection mortality rate of 6.3 per cent. This figure compares very favorably with those found in the literature. Laufman had a resection mortality rate of 15.8 per cent, Eliason and Welty[2] reported a 19 per cent mortality rate, and Koontz[4] quoted a 37.5 per cent resection mortality rate (table X).

Laufman reported that resection of less than 30 cm. of bowel has a 9 per cent mortality rate, whereas resection of greater than 30 cm. has a mortality rate of 31 per cent. The length of bowel resected in patients in this series, varied from 3 cm. to 162 cm. The only resection death was from a resection of 24 cm. of the small bowel. There were 8 cases with segments less than 20 cm. in length resected with no mortality and 8 cases with greater than 20 cm. resected with a 12.5 per cent mortality rate. It would seem, therefore, that the length of bowel resected played no significant role in influencing the mortality rate in this series.

There were relatively few complications following bowel resection. There was 1 case of intestinal obstruction and wound abscess occurring two weeks after bowel resection and a total of 4 postoperative wound infections, which constitutes a significant 25 per cent occurrence of wound infection following bowel resection.

There were 71 definitive surgical procedures in this series of 74 cases of strangulated hernia. As has been mentioned previously, 3 patients did not come to operation before death. There were 5 operative deaths, or an operative mortality of 7 per cent. There was a total hospital mortality rate of 10.8 per cent. Laufman reported an over-all mortality rate of 12 per cent; Koontz recorded a

TABLE XI

Comparative mortality rates in strangulated hernia

Confederate Memorial Hospital	10.8%
Michael Reese Hospital	12%
Johns Hopkins Hospital	10.8% (femoral only)
Mayo Clinic	23.1% (femoral only)

10.8 per cent over-all mortality rate in strangulated femoral hernias; and Jarboe,[3] in his report on strangulated femoral hernias, had a 23.1 per cent over-all mortality rate (table XI).

The operative deaths are reported briefly as follows:

Case 1. A Negro woman in the seventh decade of life was admitted to the hospital with signs and symptoms of intestinal obstruction of two days duration. She was a patient who had hypertensive cardiovascular disease and who had not taken her digitalis for one week prior to admission. In addition to the findings of intestinal obstruction an umbilical mass was found on physical examination. Approximately 12 hours after admission, while being prepared for operation, she developed profound shock. She did not respond to fluids, blood, and rapid digitalization and had to be operated upon in shock. The umbilical hernia was repaired and 24 cm. of small bowel was resected. She remained in shock throughout the procedure and died two hours postoperatively.

Case 2. A Negro woman in the fifth decade of life was admitted to the hospital to medical service with a history of four months vomiting. A Miller-Abbott tube was used intermittently for one month with the patient continuing to vomit each time the tube was removed. Roentgenologic studies were reported repeatedly as showing no evidence of obstruction. She finally was transferred to the surgical service and immediately explored. She had an internal hernia containing multiple loops of cyanotic small bowel. She developed shock at the end of the procedure. A right hydrothorax was discovered and removed by thoracentesis. She failed to respond to supportive measures and died before leaving the operating room.

Case 3. A white woman in the fifth decade of life was admitted to the hospital with an umbilical hernia that had been incarcerated for three days. The hernia was supposed to have been temporarily reduced in the admitting room by use of taxis. At operation a hole 3 cm. in diameter was found in the transverse colon with a massive fecal peritonitis. The colon was exteriorized. The patient developed shock in the operating room and remained in shock postoperatively in spite of blood transfusions. She died 10 hours later.

Case 4. A white woman in the eighth decade of life was admitted to the hospital to medical service with a history of a left groin mass appearing at times for 30 years. She also was a chronic invalid and had been bedridden with arthritis for some time. She had a five day history of intestinal obstruction and findings compatible with this diagnosis. A surgical consultant believed her groin mass to be a lipoma. She was explored seven hours later after having received only 500 cc. of physiologic sodium chloride solution preoperatively. A femoral hernia was found with a green spot on the contained loop of small bowel when it was removed from the hernia, but resection was not judged necessary. On the first postoperative day she complained of pain in the left chest and a roentgenogram of the chest revealed an infiltration of the left lower lobe. Tube feedings were begun on the second postoperative day when the patient refused to eat. She developed diarrhea and on the third postoperative day developed labored respirations, went into shock, and died.

Case 5. A Negro woman in the sixth decade of life was admitted to the hospital with a history of an incarcerated umbilical hernia for the past four years. She had developed pain in the hernia two days prior to admission but had had no obstructive symptoms. She was observed because of inconclusive findings and was not explored until three days later. At

operation an edematous transverse colon was found in the hernia. On the first postoperative day she developed chest pain and an electrocardiograph was read as showing evidence of a pulmonary embolus. She then was subjected to a bilateral superficial femoral vein ligation but died four hours after this procedure.

The nonoperative hospital deaths are as follows:

Case 1. A Negro woman in the seventh decade of life was admitted to the hospital with a strangulated right inguinal hernia and a history of eight days of fecal vomiting. Inadvertently she was admitted to the medical service and was not transferred to the surgical service until the next day. She was in shock when she arrived on the surgical ward and in spite of intensive supportive measures she died in a short time. At autopsy she was found to have a ruptured ileum with a generalized peritonitis, as a result of bowel strangulation in the right inguinal hernia.

Case 2. A Negro man in the ninth decade of life was admitted to the hospital with a strangulated right inguinal hernia of five days duration. His heart was fibrillating, he had air under the diaphragm and had the typical findings of generalized peritonitis. He remained in shock in spite of intensive measures from the time of admission until he died 24 hours later. This same patient had had a strangulated left inguinal hernia which was managed successfully at this hospital three years previously.

Case 3. A white woman in the eighth decade of life was one week postoperative from a hip nailing. She began having signs and symptoms of bowel obstruction and roentgenograms showed what was thought to be large bowel dilatation. Several surgical consultants favored a diagnosis of fecal impaction and the patient was managed with suction, enemas and fluids. She put out a large quantity of fluid per suction and soon became a severe electrolyte problem in spite of close supervision and intensive treatment. She steadily became worse and died three days later. At autopsy a strangulated femoral hernia with no obvious external mass was found.

The lessons to be learned from these cases are the usual ones in dealing with strangulated hernias and are as follows:

1. Hernias must be kept in mind constantly when obstruction is suspected and the femoral areas should be examined carefully.
2. Patients with strangulated hernias must be explored promptly after adequate preoperative fluid and electrolyte adjustment in so far as possible.
3. Attempts at reduction of incarcerated hernias by taxis are extremely dangerous.
4. Patients often need postoperative blood transfusions to avoid delayed shock as previously mentioned in this paper.
5. All hernias should be repaired electively as soon as the patient presents himself because of the dangers of later strangulation.

SUMMARY AND CONCLUSIONS

The following points have been noted in this survey:

A substantial proportion of hernioplasties done at Confederate Memorial Medical Center are for strangulation. This has been particularly true with femoral hernias where 44.4 per cent of the hernioplasties were done for strangulation cases.

Duration of symptoms has no relation to the possible viability of the strangulated bowel.

Duration of symptoms has a direct relationship to the mortality rate.

Age influences the mortality rate slightly.

Sex in this series shows that females have a predominately higher mortality rate in strangulated hernia, particularly in proportion to the number of hernias seen in females.

The mortality rate in this series compares favorably with mortality rates at other medical centers.

The condition of the bowel at operation influences the mortality rate to some extent.

The length of bowel resected does not seem to play any role in influencing the mortality rate.

There was a 25 per cent incidence of wound infection following bowel resection.

This series had a 7 per cent operative mortality rate.

There was a 6.3 per cent mortality rate from bowel resection.

There was a 10.8 per cent total mortality rate for strangulated hernias in this series.

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HYPERPARATHYROIDISM

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INTRODUCTION

Hyperparathyroidism is of particular interest to surgeons because surgery offers the only successful method of treatment. Although not a common disease, it probably is much more frequent than has been previously supposed, over 700 cases[11] having been reported in the medical literature. The syndrome usually is the result of parathyroid adenoma, but may be produced infrequently by primary hyperplasia or by carcinoma of the parathyroids.

HISTORICAL ASPECTS

In 1880, Iva Sandstrom,[20] of Sweden, first described the parathyroid glands after dissecting a series of cadavers, and five years later they were rediscovered independently by Gley. In 1890, Von Recklinghausen[22] reported 16 cases of bone disease found at autopsy, and of these, 3 were probably examples of the disease which now bear his name, "Osteitis Fibrosa Cystica of Von Recklinghausen." Fuller Albright,[1] after reexamining the findings of these cases, decided that 1 of these was hyperparathyroidism, the first described in medical literature. This case, at autopsy, showed widespread fibrosis of the bone with cysts, and giant cell tumors, and the original autopsy report re-examined by Jung[13] in 1933 reported, "Above the thyroid gland, a lymph gland, red-brown in color, is present." This description strongly suggests the presence of a parathyroid adenoma.

Erdheim,[7] in 1906, removed the parathyroid glands in rats, and thereby discovered the relationship between calcium metabolism and the parathyroid glands.

Hanson[10] and Collip[5] independently, in 1924 and 1925, isolated the parathyroid hormone and reported the elevation of serum calcium above the normal level after an injection of parathormone in dogs.

In October of 1924 Felix Mandl[16] first tried Collip's parathormone on a patient with osteoporosis and cysts of the bones. The patient was not benefited. He next transplanted fresh, healthy parathyroid glands into the patient's abdominal wall, likewise without benefit. Finally in June 1925, he operated upon the patient and removed a parathyroid tumor. Within six days the calcium disappeared from the urine and the patient was much improved. This was the first report of a case with successful removal of a parathyroid tumor.

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The first operation for this condition in this country was done in Boston, but no tumor was found. Six years later, at the seventh operation, Churchill and Cope[4, 16] discovered an adenoma in the anterior mediastinum, which was removed. The patient died 26 hours after operation.

In 1924, Dr. Charles Richardson,[19] of Macon, Georgia, removed a parathyroid adenoma successfully. This, unfortunately, was not reported in literature and did not come to light until some years later during a discussion following a paper on the subject before the Southern Surgical Association.

CLINICAL ASPECTS

Hyperparathyroidism may be divided into primary and secondary types. Nearly all of the primary types are due to parathyroid adenomas, rarely to carcinomas.

Secondary hyperparathyroidism usually is due to impaired renal function and is compensatory in nature. The currently accepted theory is that in patients with poor renal function, phosphate is not excreted in normal quantities and its concentration rises in the body fluids. The rise in the phosphate ion depresses the calcium ion to low levels, and this, in turn, calls forth a compensatory overactivity of the parathyroid glands in an attempt to maintain the normal calcium level.[6]

PHYSIOLOGY—MODE OF ACTION

Two schools of thought exist concerning the action of the parathyroid hormone. One school believes that the hormone acts directly on the bone causing dissolution and secondary electrolyte changes. Albright and his group believe that the hormone acts on the electrolyte equilibrium of the body fluids and that bone changes, when present, are secondary to the chemical changes.

The phosphate ion is believed to be affected by the parathyroid hormone in such a way that it is more easily excreted by the kidney. This produces a fall in the serum phosphorus level which, in turn, makes the body fluids less saturated. Reabsorption of calcium and phosphate from the bone occurs, thus increasing the calcium level and maintaining the equilibrium constant. As long as the calcium intake is sufficient no bone changes need develop.[2]

EMBRYOLOGY

Embryologically the parathyroid glands develop with the thyroid and thymus from two distinct primordia, the third and fourth branchial pouches. The paired parathyroid structures develop as a thickeneing of the entoderm, located between the middle layers of the deep cervical fascia. One pair is derived from the third branchial pouch with the thymus and these migrate down into the neck or mediastinum to form the inferior parathyroids. The upper parathyroid glands develop with the thyroid from the fourth branchial pouch. Supernumerary parathyroids apparently arise by division of a single parathyroid gland during its development.[12]

Lahey[14] found that the most constant position for the inferior parathyroids is close to the entrance of the inferior thyroid artery, but they may be located

almost anywhere in the neck or mediastinum. Rarely one may be found within the thyroid itself. Black[3] stated that the upper parathyroids are well posterior, lying on the esophagus or pharynx definitely dorsal to the recurrent nerve and inferior thyroid artery and at the junction of the upper and middle third of the thyroid lobe. Those that descend into the mediastinum carry their own blood supply with them, and in such cases there is a definite vascular pedicle extending from the gland to the vessels of the thyroid. This branch usually arises from the inferior thyroid artery.

Autopsy material in 527 cases reviewed by Gilmour and Martin[9] showed 80 per cent had four parathyroid glands, 14 per cent had more than four, and 6 per cent less than four glands. Normal parathyroid glands vary considerably in size and are usually from about 6 to 8 mm. in length, 3 to 4 mm. in width, and 1 to 2 mm. in thickness. They weigh normally from 20 to 50 mg.

SIGNS AND SYMPTOMS

There are three types of cases clinically; first, those in which the predominant picture relates to the skeletal system with skeletal deformities, bone pain, pathologic fractures, and bone cysts. Second, those in which urinary tract disease is the outstanding feature. This usually occurs in the form of renal or ureteral stones, with or without infection. Third, symptoms due to hypercalcemia itself with muscle weakness, poor muscle tone, anorexia, and constipation.

In the earlier cases of hyperparathyroidism due to parathyroid adenomas the outstanding clinical features were those pertaining to the bones, and it was not until 1932 that the significance of urinary tract disease and symptoms due to hypercalcemia per se were given proper emphasis. At present it appears probable that more cases of this disease are diagnosed as the result of the finding of renal disease, particularly stones, than by any other signs.

The principle findings in the osseous system are generalized decalcification, the presence of bone cysts, and the presence of giant cell tumors. These bony findings are often spoken of as *osteitis fibrosa cystica*. The bone tumors consist of solid masses of soft tissue without bone but showing osteoblasts and osteoclasts. Bone pain is an early symptom and later bone tenderness is a constant finding. Kyphosis, scoliosis, and other deformities frequently are present, and pathologic fractures are common.

Bone tumors and cysts are more common in the jaw, the metacarpals, and the ends of the long bones. The skull may develop a ground glass appearance and dental roentgenograms reveal the disappearance of the lamina dura. However, the teeth themselves are not decalcified. Recently a new sign[8] has been described—the so-called watermelon sign. According to Fender percussion of the head of a patient with hyperparathyroidism evokes a booming, low pitched note similar to the sound made by tapping on a watermelon, in contrast to the high pitched crack evoked from a normal skull.

Urinary tract findings consist of renal and ureteral stones and frequently infection of the kidneys. Polydipsia, polyuria, and nocturia are frequently present. These manifestations are due probably to a hypercalcemia, hypercalcuria, and hyperphosphaturia.

Precipitation of calcium phosphate in the convulated tubules and interstitial tissues of the kidney may lead to nephrocalcinosis. Function of the tubules is impaired, first with resulting failure of the concentration of urine, while the glomerular function, or filtration, remains normal until late in the disease.[21] Stones form as the result of the hypersaturation of the urine with calcium, and since there is always an excess of phosphate ions, the calcium is likely to precipitate out as the phosphate salt, especially in an alkaline urine, in which phosphates are insoluble. When urea splitting organisms are present in the bladder, ammonia is produced and the urine becomes alkaline. When oxalates and acid urine are present, calcium oxalate stones will form.[17] Rienhoff[19] has pointed out that whenever attention is directed to this syndrome by renal manifestations there will be a great increase in the frequency of diagnosis of the disease, since the most common complication of hyperparathyroidism is some form of renal pathology. Surely any patient who repeatedly forms urinary tract stones should be investigated for the possibility of hyperparathyroidism. In the Massachusetts General Hospital series urinary tract manifestations were present in 52 of 64 cases, while bone disease was present in only 35.

The third manifestation of hyperparathyroidism is that group of symptoms due to hypercalcemia per se. These symptoms consist of vague, nonspecific complaints of muscular weakness, constipation, and poor appetite. They probably are caused by poor muscle tone of both smooth and striated muscles. High serum calcium blood levels cause a decrease in excitability of the nerve muscle apparatus, and this is in direct contrast to tetany, where there is an increased excitability. The electrocardiograph may show a shortening of the Q-T interval, which is a measurement of the ventricular systole. Anemia may be present, but this probably is due to urinary tract infection rather than to any bone changes as a result of the disease.

LABORATORY FINDINGS

Although the diagnosis of hyperparathyroidism should be suspected from signs and symptoms, it is the laboratory and the roentgenologic examinations which make the actual diagnosis. The typical laboratory findings consist of a high serum calcium, and a low serum phosphorus. One must also obtain an alkaline phosphatase level, blood urea nitrogen and total protein determination. Urinary calcium excretion measured by the Sulkowitch test is of great value as a screening procedure. It indicates not only the presence or absence of calcium but gives a rough idea of the quantity present.

Because a portion of the total serum calcium is combined with the protein in the form of calcium proteinate, it is necessary to determine if the total serum protein is normal. If it is low, it is possible the serum calcium might give a false normal value, while if the total serum protein is high, a falsely elevated calcium blood level would be reported. McLean and Hastings[15] devised a nomogram which is an aid in evaluating the serum ionized calcium level with reference to the serum protein level.

In the evaluation of the serum phosphorus the renal function is important because phosphorus may be retained and falsely elevated to within normal

limits. The blood urea nitrogen determination is helpful in evaluating this problem.

Marshall,[17] of the Lahey Clinic, reported that depression of the serum phosphorus level is not as consistent as elevation of the serum calcium level. Fifteen of 22 patients had phosphorus levels below normal and in 7 other patients the levels were normal.

Serum alkaline phosphatase is a measure only of bone destruction or formation, and it may be within normal limits in severe cases of hyperparathyroidism. Pugh,[18] at the Mayo Clinic, states that if the alkaline phosphatase is normal there is no chance that roentgenograms will show any bone changes.

Excretion of calcium in the urine may be tested in a simple manner by the Sulkowitz test. It also is a great value postoperatively. A normal urinary calcium excretion is 100 mg. per day. Over 200 mg. per day is believed to be diagnostic of hyperparathyroidism.

SURGICAL TREATMENT

The only treatment for hyperparathyroidism is surgical. Preoperatively it is important to avoid a high calcium diet as this may precipitate calcium in the urinary tract and cause severe renal damage.

The exploration of the neck is done thorugh a collar incision. It is important to extensively mobilize the lateral lobes of the thyroid for better access to the pharyngoesophageal fold. The immediate aim at operation is to find four parathyroid glands. Black[3] pointed out that the responsibility for curing this potentially fatal disease rests largely with the surgeon who does the first operation, because of hemorrhage and scarring, which makes subsequent surgery extremely difficult in the identification of the glands. Careful hemostasis is important because blood obscures the characteristic red-brown color of the parathyroid tissue. The dissection must be meticulous.

It is imperative to have the services of a pathologist to interpret a frozen section of material suspected of being parathyroid tissue during the course of the operation. Cope[6] pointed out that it is important not to remove normal parathyroid tissue.

In cases of secondary hyperparathyroidism, 200 mg. of tissue are left behind to preserve normal calcium and phosphorus metabolism. Of 733 cases reported in the literature, 691 were due to adenoma, 28 to hyperplasia of all parathyroid glands, and 14 to cancer.

There is a rough correlation between the intensity of the disease process and the size of the adenoma, the larger the adenoma the more severe the disease.

POSTOPERATIVE CARE

The complication most likely to arise following the removal of a functioning adenoma is hypocalcemic tetany. The normal parathyroids often will have atrophied in the presence of the excessive parathormone secretion of the tumor, and will require time to regain their normal function. The patient must be supported in the meantime by substitution therapy.

Immediately postoperative the patient can be followed closely by serum cal-

cium determinations, as well as frequent Sulkowitch tests for urinary calcium excretion. If none is being excreted, it is an indication to increase the calcium intake either orally or parenterally.

As has been previously mentioned, oliguria occurs immediately postoperatively, and intravenous fluids should be given to prevent precipitation of calcium in the tubules. Development of oliguria is an immediate indication that the tumor has been removed.

Calcium lactate is given by mouth, augmented by A.T. 10 (dihydro-tachysterol) or by massive doses of vitamin D. Parathormone can be used for the first 48 hours until the A.T. 10 becomes effective. If there has been extensive skeletal disease, and especially if the alkaline phosphatase has been elevated above 20 Bodansky units, severe tetany is to be expected postoperatively.

CASE REPORTS

We report 4 cases of this disease. As far as we have been able to determine, these are the only cases in which the diagnosis has been proved and the patient operated upon in this area.

The first case is that of a 28 year old white woman, R. M., who was first seen in the emergency room at Holy Cross Hospital in January 1953. She was acutely ill and complaining of severe left flank pain with dysurea, burning, and frequency. Her past history revealed that at the age of 14 years she had had osteomyelitis involving her right knee, which necessitated several operations over the next four years. When she was 19 years of age she fell and sustained a fracture of the left hip. It was pinned with a Smith-Peterson nail and extension plate, and it was at this time that the patient noted the onset of her other skeletal deformities.

On physical examination the patient was acutely ill and in acute distress. She appeared 10 to 15 years older than her stated age of 28. The most striking physical findings were the skeletal deformities—severe kyphosis, scoliosis and chest deformity. The right leg was 2 inches shorter than the left, and the right knee was limited in motion to about 30 degrees. There was acute pain to palpation over the left flank and groin region. Figures 1 and 2 demonstrate more clearly than words the extent of her deformities.

On admission her blood study showed a white blood cell count of 8,000, per cu. mm., a hematocrit 41 mm., and a blood urea nitrogen of 12.8 mg. per cent. Urinalysis showed specific gravity of 1.020, albumin 2 plus, and urine loaded with white cells. Intravenous pyelograms showed good function of the right kidney but no excretion on the left. A calcified density was noted at the approximate position of the distal end of the left ureter (fig. 3). A left nephrectomy was done for a large hydronephrotic kidney with a suppurative pyelonephritis and abscess formation.

Two weeks after her discharge from the hospital she was readmitted with a 24 hour history of severe left upper quadrant pain, nausea and vomiting. On this occasion a diagnosis of pancreatitis and a nonfunctioning gallbladder were made on the basis of radiographic findings. She had serum amylase of 335 Bodansky units and jaundice. These signs subsided without surgery.

FIG. 1

FIG. 2

FIGS. 1, 2. These pictures were taken one year after she was first seen. The large scars on her right leg are from her previous osteomyelitis. The chest and back deformities are well visualized, as well as her scoliosis.

Four weeks later she was again admitted, this time for a thrombophlebitis of the left leg. Roentgenograms of the pelvis on this admission showed a possible destructive, sclerosing lesion of the left sacroiliac joint. Repeat roentgenograms of the pelvis again showed the destructive lesion of the left sacroiliac joint and, in addition, multiple destructive areas were seen throughout the pelvis and upper end of the left femur (figure 4). Skeletal survey showed marked osteoporosis (figure 5) with multiple cystic areas in both humeri, right ulna, proximal end of the left femur, distal end of the right femur, and the entire right tibia. Dental films (figure 6) showed the lamina dura to be nearly absent. Absence of the lamina dura and cortical absorption as shown on figures 4 and 6 are pathognomonic of hyperparathyroidism. Laboratory work showed a hematocrit 40 mm., serum calcium 12.5 mg. per cent, serum phosphorus 1.1 mg. per cent, and alkaline phosphatase 15.8 Bodansky units. Urinalysis showed the urine to be loaded with white cells and a urine Sulkowitch was 4 plus.

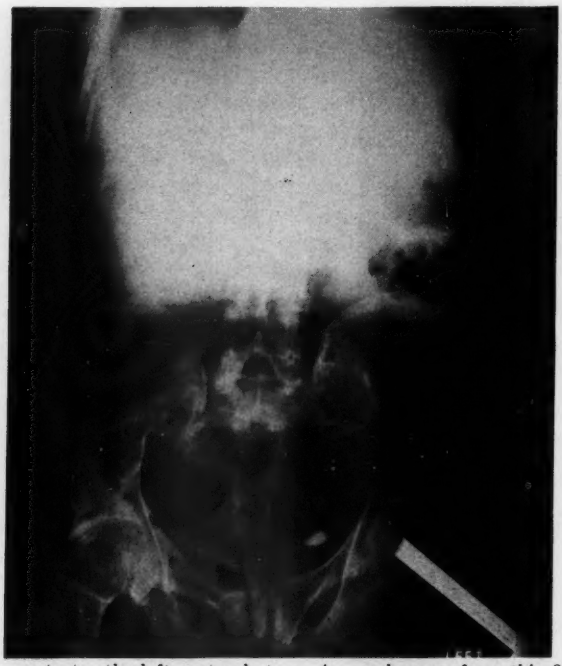

FIG. 3. Demonstrates the left ureteral stone. As can be seen from this film, the chest cage practically rides on the ilial wings. There was only a distance of about 10 cm between the xiphoid and pubis.

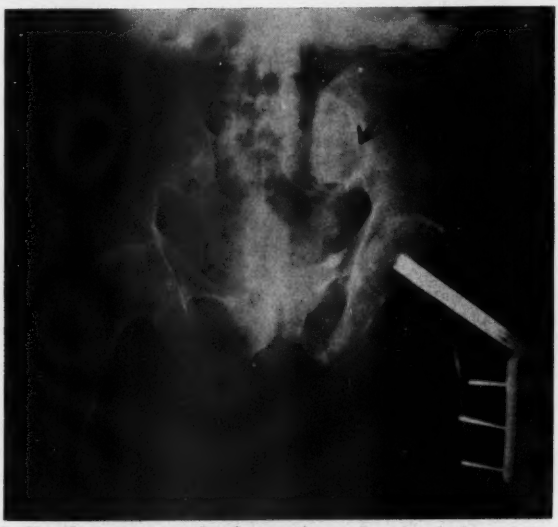

FIG. 4. This film demonstrates the destructive, sclerosing lesion of the left sacroiliac joint. A destructive lesion is present in the right pubic ramus. An area of cortical absorption can be seen about the end of the screw in the left femur.

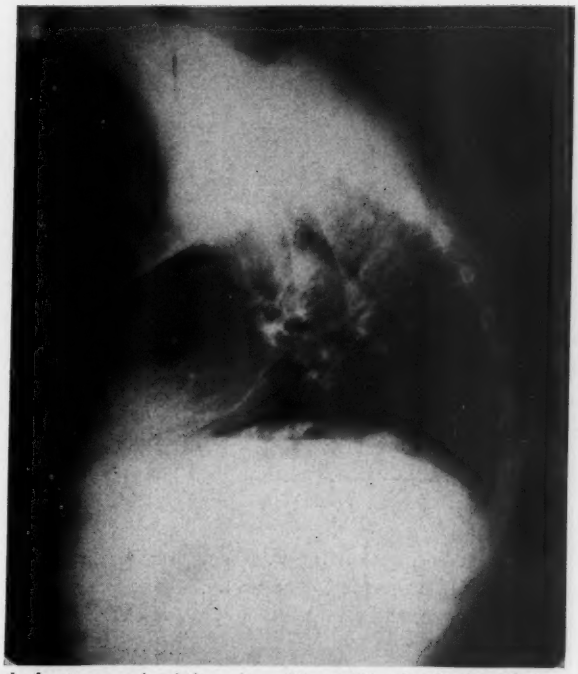

FIG. 5. Marked osteoporosis of the spine with resulting kyphosis and chest deformity

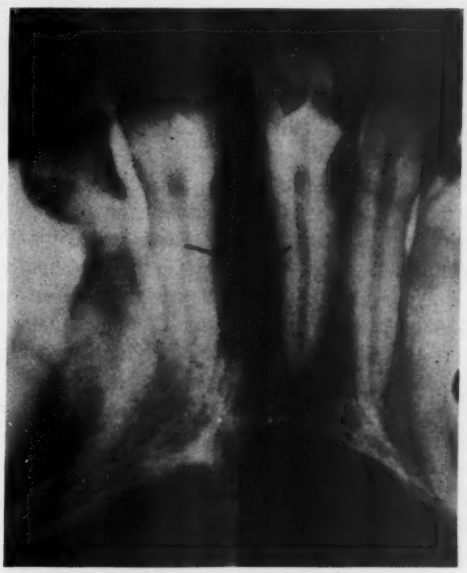

FIG. 6. Dental films showing the lamina dura to be nearly absent

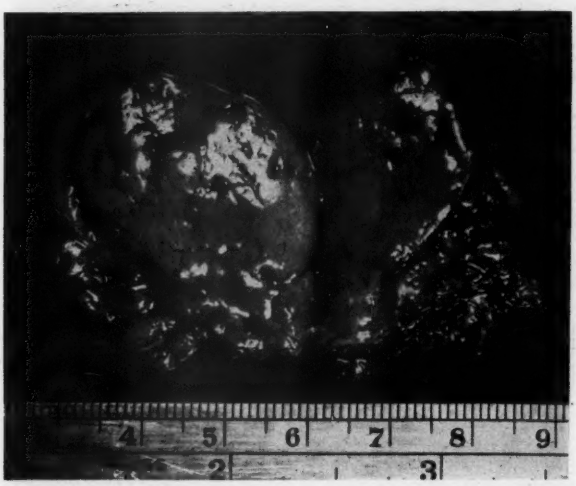

FIG. 7. Parathyroid adenoma measuring 3.5 by 3 by 2 cm. Weight 15 Gm.

It was thought that, because of the persistent pyuria, the remaining portion of the left ureter should be removed. The ureter was found to be about 2.5 cm. in diameter, filled with pus, and a large stone was present at the distal end. The patient recovered rapidly.

Eleven days later she was operated upon for the removal of a parathyroid adenoma. The thyroid was mobilized extensively. Numerous nodules were biopsied by frozen section but were reported as normal thyroid. Some bleeding was encountered at the right middle thyroid vein and this area had to be packed. While waiting for the bleeding to subside, the sternum was split and a search for the tumor was made in the superior mediastinum, but none was found. While removing the packs on the right side of the thyroid a small quantity of necrotic tissue was noted at the site of bleeding. This area was explored further and a large tumor mass was found deep behind the carotid and right subclavian arteries and adjacent to the trachea and esophagus.

The adenoma reasured 3.5 by 3 by 2 cm. and weighed a little over 15 gm. It was brick-orange in color, very soft and friable. The histologic picture was that of a chief cell tumor in which the cells had become acidophillic. Figure 7, 8 and 9 demonstrate the tumor. As can be seen there is a homogenous appearance to the cut surface of the adenoma and it appears highly cellular. The histologic picture shows the cords of chief cells.

The date sheet (table 1) shows the patient's postoperative course and follow up to the present time, with the changes due to treatment. It also compares the laboratory work of the other 3 patients. Because of the degree of skeletal decalcification, it has been necessary to carry her on high oral doses of calcium and vitamin D. She performs her own Sulkowitch test at home, thereby helping to regulate her calcium intake and prevent over-dosage.

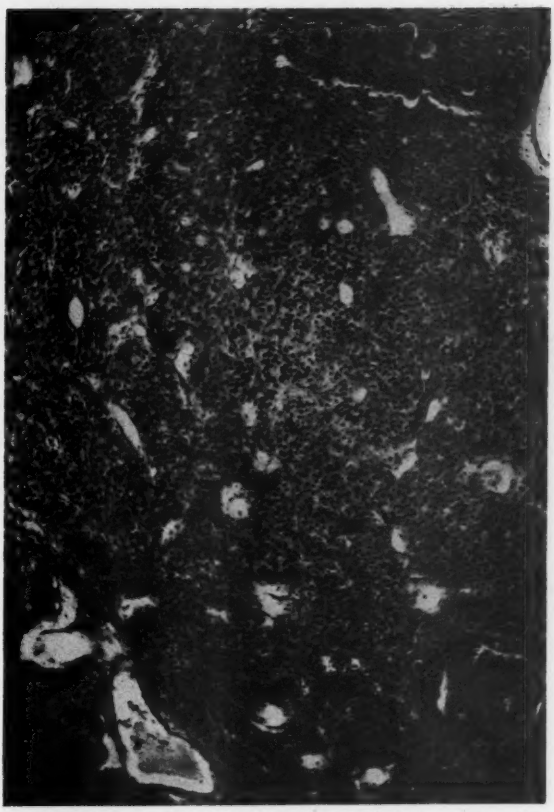

FIG. 8. Low power microscopic view of tumor showing the cords of chief cells about sinusoids.

She has done well postoperatively and kidney studies at the present show the right kidney to be functioning without impairment; she has had no further pyuria. Figure 10 shows the bony changes which have occurred since the removal of the adenoma. The bone trabeculations have been re-established and the cysts have become less apparent.

The other 3 cases will be considered only briefly. The second case was a 26 year old white woman, M. T., mother of three children, reported through the courtesy of Dr. Philip B. Price. She was seen first at the Salt Lake County Hospital in August 1952, at which time she delivered a full term child. She was readmitted one month later with acute right flank pain, urinary frequency and microscopic hematuria. She gave a history of recurrent renal colic since her first pregnancy. Two years before she was seen at Salt Lake General Hospital, a stone was removed for the left renal pelvis. During the last pregnancy she had had two operations for renal stones. On admission the hematocrit was 41, white

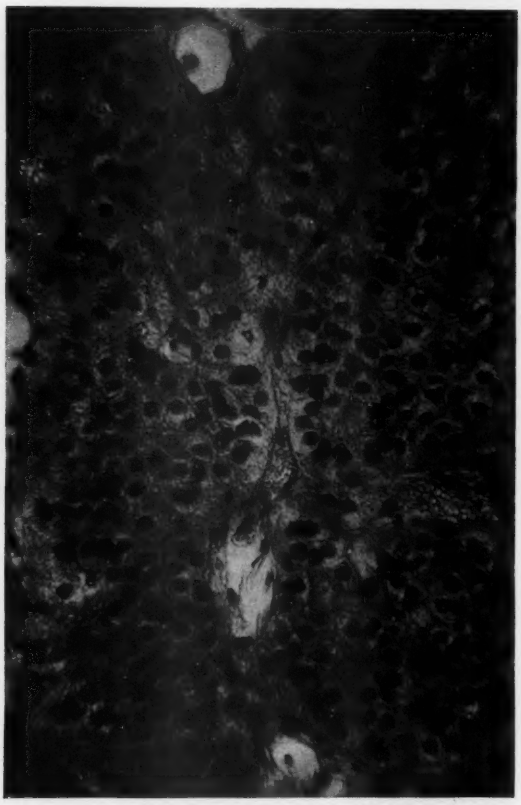

FIG. 9. High power microscopic view showing the uniform cells with a clear cytoplasm. This is characteristic of a chief cell tumor.

blood cells 12,000 per cu. mm., blood urea nitrogee 40 mg. per cent, serum calcium 12 mg. per cent, serum phosphorus 6.5 mg. per cent. Studies showed stones in both kidneys. A right pyelotomy was done because of obstruction to the right ureter. She was treated later in the Out-Patient Department for the kidney condition, and in August 1953, was readmitted for the study of possible hyperparathyroidism. Roentgenologic examination showed generalized osteoporosis, but no bone cysts were found and the lamina dura was present. She was operated upon and a 4 by 3 by 2 cm. adenoma was found on the right side between the carotid and the esophagus. The adenoma weighed 4.5 gm. after fixation. Microscopically this tumor was a mixed cell type. Considerable difficulty also was encountered in finding this adenoma at operation. Two months later a right ureteral calculus was removed. Prognosis in this case is guarded because of the renal damage.

The third case is reported through the courtesy of Dr. M. C. Lindem and Dr. Philip M. Howard. She was a 20 year old white woman, Miss Z., who was seen

TABLE I

Hyperthyroidism

Date	Serum Calcium mg%	Serum Phosphorus mg%	Alkaline Phosphatase Bodanski Units	Urine Sulkowitch	Therapy
R. M. 28 year old woman					
6-20-53	12.6	1.8	20.5	++++	
6-29 operated				++++−	Cal. IV 2.0 gm.
6-30	9.7			+	Cal. IV 2.0 gm.
upon					
7-1	9.0			±	Cal. IV 5.0 Gm.
7-2	8.3			−	Cal. IV 5.0 Gm. AT-10 1.25 mg. qd.
7-3	8.4	1.8	13.0	+	Cal. IV 4.0 Gm. Vit. D 6,000 u. qd.
7-8 discharged	10.5	1.6	29.7	++	AT-10 1.25 mg. qod. Oral cal. 30 Gm. qd.
7-21	9.2	2.4		±	AT-10 1.25 mg. q3d Vit. D 200,000 u. qd.
8-18	12.2			++++	AT-10 1.25 mg. discontinued
9-15	9.2	1.8	3.2	+	Vit. D 150,000 u. qd.
10-13	8.3	1.6	4.1	±	
12-8	10.0			+	Oral cal. 15 Gm. qd.
1-22-54	9.8	1.8	7.8		
M. T. 26 year old woman					
10-7-52	13.7	4.0			
2-11-53	14.0	2.3	10.2		
8-21 operated	12.4	1.8	6.5	++++	
11-18	9.2	4.7	7.3	+	
Miss Z. 20 year old woman					
1942	12.5	2.1	5.6		
M. H. 29 year old woman					
1933	14.8	6.45			

in March 1943, with listlessness, vague indigestion, and aching in the left knee, which she had had for several months. She had been hospitalized several times since the age of 14 years because of nausea, vomiting, and *acidosis*. She always had been anemic and frail. The year before a cyst and two molar teeth had been removed from the right lower mandible. Pathologic report showed this to be an epulis.

Roentgenograms of the patient's left knee showed cyst-like areas in the lower end of the femur and proximal end of the tibia. She was hospitalized at St. Mark's Hospital, where further roentgenograms showed generalized osteoporosis, cysts of the proximal and distal end of the left femur, upper end of the right

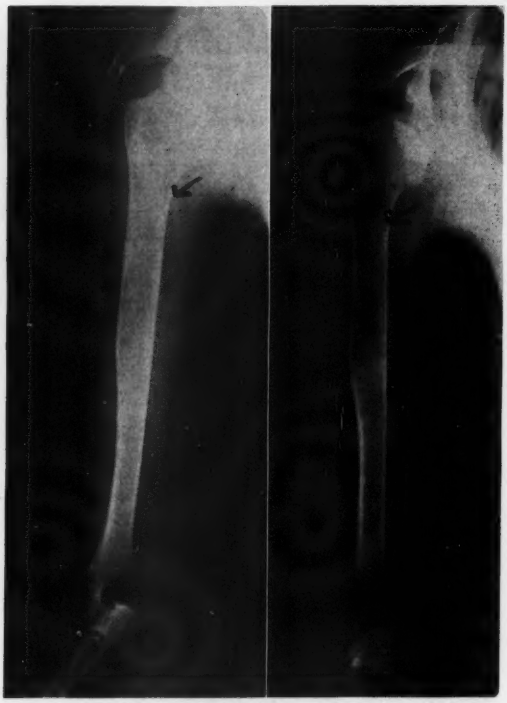

FIG. 10. Demonstrates the changes which have occurred in the seven months after removal of the tumor. On the right, a large cyst can be seen in the upper end of the right humerus and in the lateral epicondyle. On the left, the cyst in the lateral epicondyle has nearly disappeared. The trabeculation of the bone is nearly normal.

humerus, and mandible. Laboratory work showed a serum calcium of 12.5 mg. per cent, serum phosphorus 2.1 mg. per cent, and alkaline phosphatase 5.6 Bodansky units. Urinalysis was negative.

At operation the thyroid was mobilized extensively. When the left lobe of the thyroid was rotated medially, a nodular growth was noted on the posterior surface of the inferior lobe. The adenoma measured 3 by 1 by 1 cm. The cut surface was yellow and brown throughout. Microscopically this was a chief cell tumor. No oxyphillic cells were seen.

The fourth case was a 29 year old woman, M. H. This is the first case which was operated upon in Salt Lake City to our knowledge, and is reported through the courtesy of Dr. Eliot Snow. She was first seen at the Salt Lake Clinic in July 1933, complaining of lumps in her fingers and deformity of the hands. She was a normal child at birth but when 6 months old she sustained a fall and shortly thereafter developed nodular deformities of the left arm and leg. At 14 months of age her fourth and fifth fingers of the left hand were caught in a closing door and this was followed by the development of nodules on these fingers. During the next few years numerous lumps appeared on the hands and feet. These grew

very gradually and at no time were they painful or red. At the age of 9 years, she fractured her right femur, which healed normally. When in high school, she jumped off a street car and injured the right ankle. A deformity developed afterwards. The above symptoms should not be interpreted as due to hyperparathyroidism and more likely are due to her enchomdromatosis. She developed in stature up to the age of 16 years, at which time she was 4 feet and 5 inches tall.

During the year before she was seen, she had noted a gradual generalized weakness. She also had developed minor bone pain. On examination she was short and had numerous nodular deformities of the extremities. The tumors of the fingers were markedly deforming and she had diffculty in bringing the fingers together. The thyroid was approximately three times normal size and a hard nodule was felt on its left side.

Laboratory work-up showed that her urine was normal. The blood calcium was 14.8 mg. per cent. The serum phosphorus was 6.45 mg. per cent. Roentgenograms showed numerous cyst-like tumors of both clavicles, both scapulas, many ribs, the entire pelvis, and all the bones of the extremities. A diagnosis of generalized osteitis fibrosis cystica was made.

On July 27, 1933, a neck exploration was done. A mass 5 by 2 cm. in size was found lying behind the left upper pole of the thyroid on the anterior carotid sheath. Histologically this was a characteristic chief cell tumor of the parathyroid. She did well until the third postoperative day when she developed signs of tetany. This was relieved by calcium gluconate given intravenously and parathormone given intramuscularly.

In 1943 (ten years later) one of the nodular tumors was removed from her hand. This was filled with a pink cartilaginous material surrounded by a thin shell of bone. The diagnosis of enchondromatosis was added. In 1945 she had a right leg amputation for osteochondrosarcoma. She died several years later of metastases.

COMMENT

The above 4 cases present considerable variation: The first patient developed early skeletal deformities and late renal complications. The second was admitted primarily with renal disease. The third presented with an epulis and bone pain, and the fourth was one of enchondromatosis with hyperparathyroidism.

It is with interest that we review these cases, because in any one of them the diagnosis could have been suspected earlier had a very simple test been run. The Sulkowitch test, a test for urine calcium, is as easily and quickly run as the test for albumin. These cases represent only the highlights of these peoples' lives. The serious incapacitation, psychologic and social problems cannot be considered in this paper. It is well to remember that these tragedies could have been presented.

CONCLUSIONS

In conclusion we can say that hyperparathyroidism is not an uncommon disease, and it is surgically curable. The triad of symptoms may be present,

either singly or in any combination. Urinary tract manifestations are the most common findings. Skeletal changes often are reversible.

It is imperative to make the diagnosis of hyperparathyroidism early, as only in this way can the patient be operated upon and the condition corrected before severe renal and bone damage occur.

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EDITORIALS

HÜRTHLE CELL TUMORS OF THE THYROID

According to the number of Hürthle cell tumors reported in the world literature up to 1945, these tumors probably would be considered rare. Since that time a rather large number have been reported. The first reference to Hürthle cell tumors of the thyroid was made by Langhans in 1907. In our own material 5 per cent of all solitary thyroid tumors are of the Hürthle cell type.

There still is a wide disagreement in the literature regarding the histogenesis of this tumor as well as to its prognosis and treatment. To mention a few theories, some thought that they developed from postbranchial remnants, ectopic parathyroid cells, from interfollicular cells of the thyroid and that they are a variant of thyroid epithelium. The last seems the most reasonable supposition. The same type of cell may be found in the thyroid of exophthalmic goiter, Hashimoto's disease and in the thyroids of patients suffering from spontaneous myxedema.

Hürthle cell tumors occur at all ages but are much more common in the fourth and fifth decades of life. They occur in females much oftener than in males. Cases have been reported in infants.

The tumors may occur as solitary nodules in an otherwise normal thyroid or may be found in pre-existing goiters of many years' duration. They may occur in nodular colloid goiters and in exophthalmic goiters. They have a grayish-brown cut surface and a distinct fibrous capsule.

The Hürthle cell is large—15 to 30 microns in diameter. It has a finely granular acidophilic cytoplasm, a pale vesicular structure and a distinct cell border. Its shape may be polyhedral, cuboidal or columnar. The arrangement of the cells may be trabecular, small alveolar, large follicular and papillary or a combination of 2 or more types may be seen. Invasion of the capsule and blood vessels may take place. Regional and distant metastases may occur. Apparently some never metastasize and those that do may be present for years without doing so.

The Hürthle cell apparently has no endocrine function. Although patients with these tumors sometimes present toxic symptoms, these are caused by the associated thyroid pathology.

Some consider all Hürthle cell tumors malignant and others consider them benign adenomas with malignant potentialities. The American Cancer Society has classified them as Hürthle cell carcinomas. Microscopic examination will not give a definite answer as to their malignancy if the structure is orderly. It is a fairly well established fact that radical surgery, which includes removal of the lymph-bearing tissue of the neck, is not necessary even though microscopic examination shows invasion of the blood vessels of the capsule. Removal of the involved lobe will effect a cure in most cases.

Of our 25 cases with Hürthle cell tumors, 88 per cent are alive and 80 per cent are without evidence of disease following lobectomy without radical neck dissection or radiation therapy.

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BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

Ciba Foundation Symposium on Hypertension: Humoral and Neurogenic Factors. Editors for the Ciba Foundation: G. E. W. WOLSTENHOLME, O.B.E., M.A., M.B., B. CH., AND MARGARET P. CAMERON, M.A., A.B., L.S. Assisted by JOAN ETHERINGTON. With 73 illustrations. Little, Brown and Company, Boston, 1954. Price $6.75.

These proceedings of the 1953 Ciba Symposium on Humoral and Neurogenic Factors in Hypertension include 21 papers written by leading research workers from different countries. Although most of the reported material has been published before, the book provides an informative survey on the status of this complex problem. The extensive discussion following each paper reflects the wide divergence of opinions on the nature and causative factors in hypertensive cardiovascular disease.

For the practical surgeon, Goldenberg's paper on diagnostic methods in pheochromocytoma should be of special interest.

K. R. REISSMANN, M.D.

Hernia: The Pathologic Anatomy of the More Common Hernias and Their Anatomic Repair. BY CHESTER B. MCVAY, M.D., PH.D., Clinical Professor of Anatomy, The University of South Dakota School of Medical Sciences; Surgeon, The Yankton Clinic, Yankton, South Dakota. Charles C. Thomas, Publisher, Springfield, Illinois, U. S. A. Price $4.75.

This recent book by Chester B. McVay is one of the Pictorial Surgical Techniques Series, published by Charles C Thomas. This book contains only 40 pages of textual and illustrative material. There are 19 plates, most of which contain multiple line drawings. These illustrations are of superb quality. The textual matter is closely correlated with the drawings.

Doctor McVay presents the common types of hernias with emphasis upon anatomic defect, and describes the methods used by him in their repair. Each technic has been used with satisfaction by the author. His results have been excellent.

The book appears to be especially valuable to the general practitioner who is faced with an occasional herniorrhaphy, or to the surgical resident. Some of the established surgeons might do well to heed some of Doctor McVay's suggestions.

T. G. ORR, JR., M.D.

Patologia del Estomago Operado, Segundo Congreso Argentino de Gastroenterologia, 1953. Sociedad de Gastroenterologia de Buenos Aires, Associacion Argentina de Radiologia, Sociedad Medica de Mendoza. Editorial Universitaria, Paraguay 2074, Buenos Aires.

This book is a symposium on the subject of "Pathology of Gastrectomy" sponsored by the Gastroenterologic Society of Buenos Aires, Argentina. Interns, surgeons and radiologists from Argentina, Uruguay, Brasil and Chile participated in this extensive study of the changes produced in the gastrointestinal tract by gastrectomy, either partial or total, for peptic ulcer or malignancies. All points are well covered, ranging from the hematologic changes and speculation about the reasons for the low incidence of pernicious anemia in total gastrectomy cases to the various radiologic aspects of gastrectomies, the immediate postoperative morbidity and the repercussions upon the biliary system. Attention is given to the diagnosis as well as to the treatment, and a special chapter is dedicated to the nutrition problems of the gastrectomized patient.

It is unfortunate that the graphic part of this book is not up to standard, particularly the radiographic pictures, but this does not detract from the high value of this book for the many interested in the problems of gastrectomy.

EVERTON M. SANTOS, M.D.

Demonstrations of Operative Surgery, A Manual for General Practitioners, Medical Students and Nurses. By HAMILTON BAILEY, F.R.C.S., F.A.C.S., F.I.C.S., F.R.S.E. Emeritus Surgeon, Royal Northern Hospital, and Consulting General Surgeon, Metropolitan Ear, Nose and Throat Hospital, London; Hunterian Professor, Royal College of Surgeons of England; formerly External Examiner in Surgery, University of Briston; and General Nursing Council. Second Edition. E. & S. Livingstone, Ltd., Edinburgh and London, 1954. The Williams & Wilkins Company, Baltimore. Price $6.00.

In his preface to this second edition, the author states that the purpose of the book is a "manual of introduction to operative surgery" for students. The first edition actually was a "verbatim report of a series of demonstrations given in the operating theater mainly for the benefit of the nursing staff and a few general practitioners taking a refresher course."

In this edition, Mr. Bailey has supplemented his book with chapters by special contributors. The volume is profusely illustrated with plates of generally good quality.

This book would be of value to a student nurse or medical student beginning their academic experience in surgery. However, it should be pointed out that some of Mr. Bailey's indications for certain procedures might be open to discussion by many surgeons. But as far as demonstrating operative and operating room procedures, the book is quite adequate.

T. G. ORR, JR., M.D.

A Stereoscopic Atlas of Human Anatomy. By DAVID L. BASSETT, M.D., Associate Professor of Anatomy, Stanford University, California. Section II, Head and Neck, Reels 35–84. Color photographs by Wm. B. Gruber. Sawyer's, Inc., Portland, Oregon. Price $38.50.

Dr. Bassett's approach to descriptive anatomy is truly a refreshing one. In this second section of what is planned to be a complete anatomical atlas, he covers the Head and Neck. Each volume contains ten "View-Master" stereo views of dissections or specimens in full color. In a foreword by C. H. Danforth, he states that the principal purpose of the atlas is to provide ready review of regional dissections when needed by either student or clinician.

For each stereo view, the text contains a very good line drawing with identifying numbers, the code for which is supplied on the facing page. The atlas contains no descriptive text. The specimen dissections are cadaver preparations of the highest quality with differential color injection of the vascular system.

At present only Sections 1 and 2 are available, covering the Central Nervous System and Head and Neck, respectively. Regional Sections on Upper Extremity, Thorax, Abdomen, Pelvis, and Lower Extremity are in preparation. We are eager to review these when available.

This unique and stimulating approach to anatomy should be valuable to anyone confronted with the necessity for anatomical review and would be an aid to the freshman student.

T. G. ORR, JR., M.D.

A History of Medicine. Two volumes. By RALPH H. MAJOR, M.D., Professor of Medicine and of the History of Medicine, University of Kansas School of Medicine, Kansas City, Kansas. Charles C Thomas, Publisher, Springfield, Illinois, U. S. A. Price $14.50.

This History of Medicine might well be called a Story of Medicine. It is not a compendium of dry facts which tires the mind and stifles the imagination, but a lively account of the fascinating events, eras, and epochs in the development of medicine from the dawn of history.

History often is dull reading. Such is not true of this history. Outstanding events in

the development of medicine are correlated with events in general history which not only makes interesting reading but serves to fix in the mind the time and the place of medical history in the history of the world.

The style of writing is fluent and clear. Such passages as 'Medicine is a vast edifice resting on a foundation of many stones', 'This generation was slowly forgetting the miseries resulting from the Napoleonic wars and from the French occupation and was recovering from the offspring of this mental and physical degradation—the romantic, mystic and soothing precepts of *Naturphilosophie*', 'He probably missed the masterly directing hand of Müller and the stimulating atmosphere breathed by Müller's pupils', and 'The two revolutionary discoveries in surgery—anesthesia and antisepsis—were made within the space of 20 years. Fate dealt quite differently with the two discoverers. Morton died embittered, discredited by many, and practically penniless. Lister died a peer of the realm, honored by the World', are examples of the writing which makes this history a story.

Since Doctor Major began the study of medicine he has been interested in its history. These volumes are the culmination of years of study, with visits to 20 countries, including the islands of Cos, Crete and Rhodes, to gather material at its source. The books contain 36 original illustrations taken by Doctor Major on his travels.

The binding, printing, and 422 illustrations of these 1153 pages of history are all that could be desired. The text is divided into epochs, beginning with the earliest records known and ending with the middle of the twentieth century. To make the story complete there is a Biographical Addenda at the end of each epoch, arranged in chronological order. These Addenda make unnecessary numerous footnotes, which are an abomination in any book.

In the preface Doctor Major states that 'This book is not written for the specialist in medical history to whom most of the facts related are already well known. It is written primarily for the medical student and medical practitioner, in an attempt to interest them more in the history of their own profession, which, despite attacks and abuse, despite quacks and charlatans, and in spite of its own mistakes, has proved itself more enduring than any of the civilizations which gave birth to it.' This statement prompts me to express the opinion that medical history should be a part of the required study of the senior year in medical school. We are required to study general history in our preparatory schools and universities for its general education and cultural values. Is it not just as important that medical students be taught the history of their profession?

Doctor Major further states: 'I have attempted to write a continuous account of the stream of medical history, punctuated with the names of eminent physicians often accompanied by a brief biography.' As I read the History of Medicine I am impressed by the number of diseases and conditions which are known by the names of the authors who described them. This is as it should be. In spite of some criticism to the contrary, the names of discoverers should not be dissociated from their discoveries. The association of great names in medicine with their contributions adds interest and stimulates the memory and admiration for those who have made our profession great. Great names cannot logically be separated from great accomplishments.

We speak glibly of anaphylaxis, vitamins, viruses, antibiotics, anaesthesia, asepsis, and many other great gifts to medicine, often without knowing that physicians and scientists spent a major portion of their lives in giving such priceless possessions to medicine and to the public. These things the student of medicine should know. The stories of these great accomplishments are in Doctor Major's books.

If one critically analyzed each epoch of medicine that Doctor Major describes, he would end the analysis with nothing but praise for the book.

I hope that this review will not be considered exaggerated. I admit freely that I may be somewhat biased in my appraisal of this History of Medicine because of my many years of association and friendship with the author. I recommend that you read the volumes and judge for yourself.

THOMAS G. ORR, M.D.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Analecta Genetica: Collana Di Monografie. Diretta Da LUIGI GEDDA. Genetica Medica. Iᵘᵐ Symposium Internationale Geneticae Medicae. 6–7 Septembris MCMLIII. Edizioni Dell'Instituto Gregorio Mendel. Roma 1954.

Cleft Palate and Speech. By MURIEL E. MORLEY, B.Sc., F.C.S.T., Speech Therapist to the Royal Victoria Infirmary, the Hospital for Sick Children, and the Newcastle General Hospital, Newcastle-upon-Tyne. Third Edition. Edinburgh, E. & S. Livingstone, Ltd., Edinburgh and London, 1954. Williams & Wilkins Co., Baltimore. Price $4.25.

Spinal Epidural Analgesia. By P. R. BROMAGE, M.B., B.S. (London), F.F.A.R.C.S., D.A. Consultant Anaesthetist, Chichester Hospitals Group; Portsmouth Hospitals Group; Ministry of Pensions Hospital, Cosham. Visiting Anesthetist, King Edward VII Sanatorium, Midhurst. Baltimore, The Williams & Wilkins Company, 1954. Price $3.75.

Hysterectomy. By JOHN C. BURCH, M.D. AND HORACE T. LAVELY, M.D. Publication Number 226, American Lecture Series. A Monograph in American Lectures in Gynecology and Obstetrics. Edited by E. C. HAMBLEN, B.S., M.D., F.A.C.S. Charles C. Thomas, Springfield, Illinois, 1954. Price $5.50.

Hugh Roy Cullen: A Story of American Opportunity. By ED. KELMAN AND THEON WRIGHT. Illustrated by Nick Eggenhoffer. Prentice-Hall, Inc., New York. Price $4.00.